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THYROXINE THERAPY IN OTOSCLEROSIS. REPORT OF 42 CASES.*

DR. MAX A. GOLDSTEIN, St. Louis.

In the summer of 1935, I had the opportunity of visiting with the late Dr. Albert A. Gray at Middlesex Hospital, in London, where he was working under a stipend of the Ferens Institute on the evaluation of thyroxine therapy, locally applied within the middle ear, as a means of determining whether improvements in hearing noted in selected otosclerotic cases could be found of sufficiently long duration to be of practical value.[1]

The basis of this therapy, as explained by Gray, in his report to the Royal Society of Medicine, in May, 1935, was "to ascertain if, by producing an active congestion in the minute blood vessels in the organ of hearing in cases of otosclerosis, an improvement in the hearing occurred, and thus testing the correctness or incorrectness of the author's view of the etiology of the disease as expressed by him in the Dalby Memorial Lecture of May, 1934."[2]

In February, 1936, I reported before the Midwest Section meeting of this Society[3] my preliminary observations on a first group of four selected cases and offered my observations on the technique as I found it applied. Since then I have proceeded with this technique and have collected data on 42 cases of selected young otosclerotics; I offer the results in this paper for your consideration.

*Read before the Forty-fourth Annual Meeting of the American Laryngological, Rhinological and Otological Society, Atlantic City, April 28, 1938.

It is true that this form of approach is rather empirical in character, and I have no physiological or biochemical explanation to offer as to the results obtained. My observations, therefore, must deal essentially *1.* with the technique of applying the thyroxine therapy; *2.* the reaction, as determined by inspection of the field of injection; *3.* the patients' reports; *4.* careful audiometric tests before and after this therapy, and *5.* the evaluation of its permanency as indicated in subsequent audiograms within six months to a year following treatment.

Gray's first purpose was "to find some agent which, when applied locally, would produce an active congestion of the blood vessels and at the same time be free from any irritating qualities which might cause an inflammatory reaction.

"By the work of Kendall, Barger and other biochemists, the active principle of the thyroid gland 'thyroxine' has been isolated and is now made synthetically. This preparation is more satisfactory for practical use than the crude thyroid extract of the gland itself."

Quoting Gray, "there is no evidence whatever that a deficiency of the natural hormone of the thyroid gland occurs in otosclerosis; and thyroxine was not selected to compensate for any such supposed deficiency. It was chosen solely on account of its local action, and any other substance which has a similar local action would do equally well, but the writer does not know of any such agent."

Thyroxine may be obtained in the form of tabloids of 1/64 gr. (0.001 gm.), this representing the official maximum dose.

I have consistently used this dosage (1/64 gr.) in my entire series of 42 cases and have scrupulously followed the technique of Gray.

The thyroxine tabloid is dissolved in four drops of warm, sterile, distilled water and is injected through the drum membrane directly into the middle ear cavity so that it may contact the promontory and oval window.

Any well-made, small hypodermic syringe with a well-fitting glass plunger will answer. The needle, as illustrated, is bayonet-bent, somewhat longer than the external auditory canal, the bevel at the tip short and stumpy, somewhat similar to that in needles used for intravenous injections.

CASE 1.

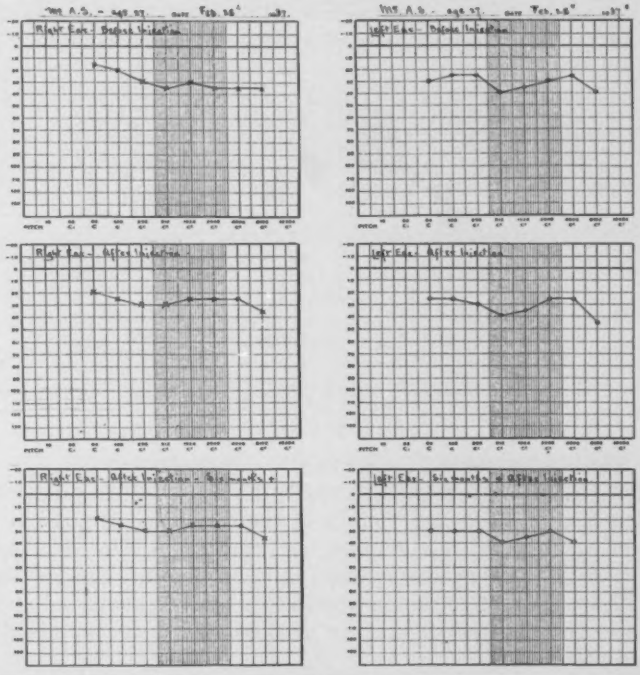
Familial History: Patient has younger sister hard-of-hearing.

Duration and Character of Deafness: Gradually increasing.

Other Symptoms: 13 mos. pulsating tinnitus, especially in L. ear.

Drum Membrane: Left presents flushed, red appearance; considerable retraction about malleus handle. Tip of malleus handle reduced in normal movement. R. ear does not show such marked evidence of impaired mobility.

Eustachian Tubes: Pharyngeal ends have same low-grade inflammation as evident in ears. Both tubes patulous.



Notes on Injection: Anesthesia perfect. Puncture at point of election. No exudate except after second injection. Pulsating tinnitus seemed louder after first injection.

Notes on Reaction: Patient reported feeling of stuffiness in L. ear after last injection.

Hearing Improvement: From 5 to 20 dcb. in both R. and L. ears.

Duration of Improvement: Slight depreciation shown in some tones but patient reports tinnitus cleared up on R. ear and lessened in L. ear.

Patient's Observation: Still has feeling of stuffiness in L. ear.

In the accompanying illustration, the simple equipment for this technique is presented (see Fig. 1).

The thyroxine tabloid is placed on a watch glass and rubbed up with four drops of sterile water with a short glass stirring rod.

One of the valuable factors in carrying out this technique is the complete anesthesia of the drum membrane. This is accomplished by the instillation of 15 to 20 drops of aniline oil-cocaine solution: (C.P. aniline oil 90 parts, cocaine hydrochlorate 10 parts). The aniline oil must be freshly prepared and should be colorless or of a very faint straw tint.

Fig. 1.

The patient's head is inclined to a completely lateral position for the ear to be anesthetized; the aniline oil-cocaine solution is dropped to the fundus of the canal and is allowed to remain there for five minutes, accurately timed by the clock. The aniline oil-cocaine solution is then carefully mopped out with pledgets of cotton on applicators until the external auditory canal and entire surface of the drum membrane is dry and clear.

It should be noted that the aniline oil penetrates the skin quickly, swelling the stomata, and thus becomes the ideal vehicle for the cocaine to penetrate as an anesthetizing agent of the drum membrane. It should also be emphasized that aniline oil may produce symptoms of aniline poisoning if it is not carefully removed within the time empirically set for its action on the drum membrane.

Let me observe at the outset that in the 168 injections made in this series of cases, complete anesthesia of the mem-

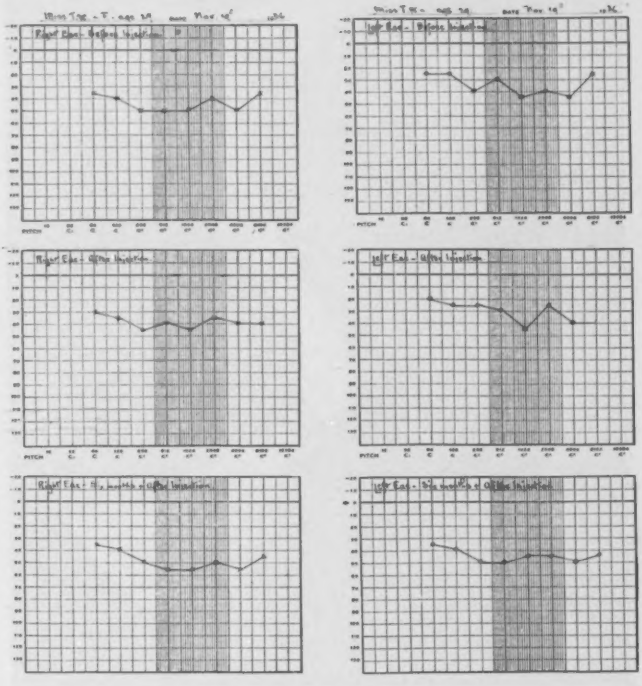
CASE 2.

Familial History: No history of deafness for several generations.

Duration and Character of Deafness: Deafness noticed over several years, gradually increasing.

Drum Membrane: Normal in color; moderate retraction; comparatively small external auditory canal.

Eustachian Tubes: Both patulous.



Notes on Injection: Anesthesia perfect. Puncture at point of election. No exudate.

Notes on Reaction: No discomfort.

Hearing Improvement: From 5 to 10 dcb. on R. ear and 5 to 15 on left throughout tone range.

Duration of Improvement: Some retrogression in L. ear. Audiogram curve in R. ear straightened out.

Patient's Observation: Speech appreciation much clearer.

brana tympanum has been obtained without exception and no untoward effects from cocaine or aniline oil observed.

We are now ready for the thyroxine injection: The point of election for the insertion of the hypodermic needle is at the point half-way between the tip of the malleus handle and the posterior rim of the annulus tympanicus (see Fig. 2).

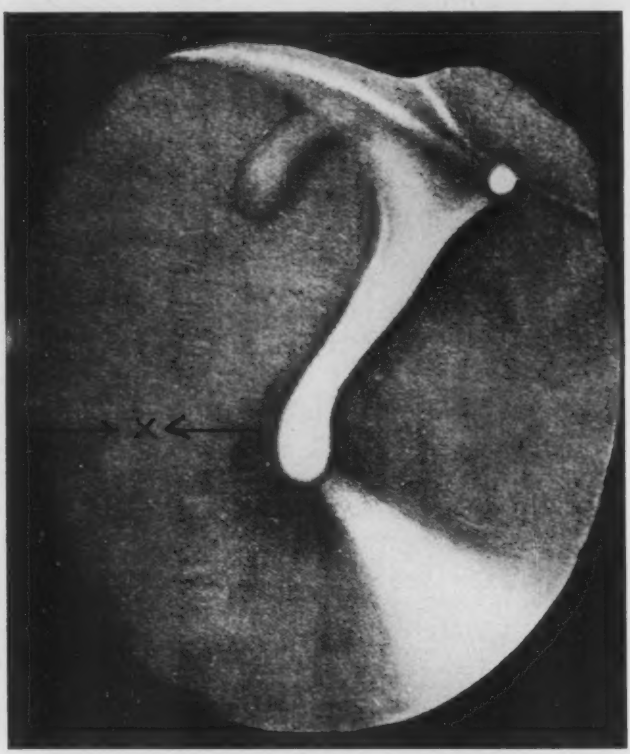


Fig. 2.

Immediately after the injection, the patient, now in an erect position and with the head thrown well backward, is directed to keep the mouth open wide for three minutes, thus preventing part of the injected fluid from escaping down the Eustachian tube. This position also insures, as far as possible, bathing the region of the round window.

Inspection of the drum membrane after injection usually shows a slight bulge along the posterior-inferior quadrant,

CASE 3.

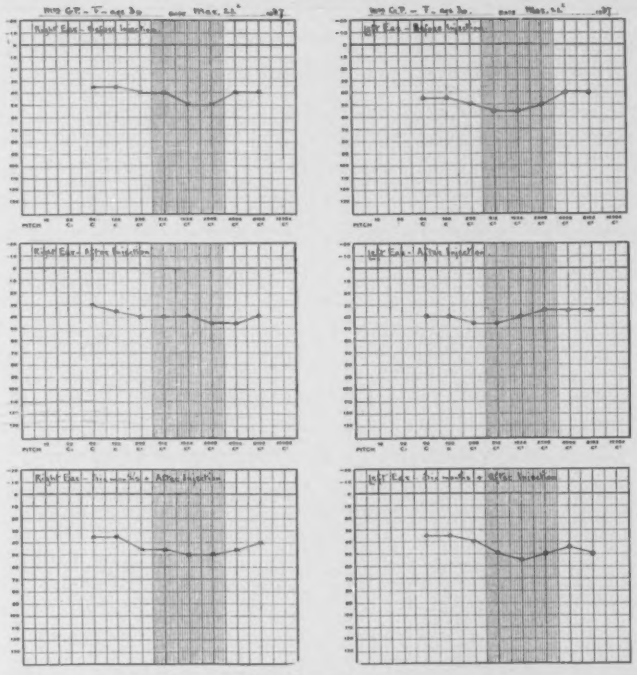
Familial History: No history of deafness for several generations.

Duration and Character of Deafness: Several years, gradually increasing in severity.

Other Symptoms: High pitch tinnitus (R. and L.), 1,024 d.v.

Drum Membrane: Normal in color and lustre, but somewhat retracted in R. ear; L. ear markedly retracted around malleus handle and surface appears "mealy."

Eustachian Tubes: Both patulous.



Notes on Injection: Anesthesia perfect. Puncture at point of election. No exudate. When making first injection in L. ear, needle seemed to meet some obstruction, but injection was made without pain or other difficulty.

Notes on Reaction: No discomfort of any kind, except feeling of fulness.

Hearing Improvement: From 5 to 15 dcb. in L. ear and from 5 to 10 dcb. in certain tones in R. ear.

Duration of Improvement: 11 mos. after injection shows improvement in some tones, others unchanged.

Patient's Observation: Hearing perception much clearer.

CASE 4.

Familial History: No record of deafness for several generations.

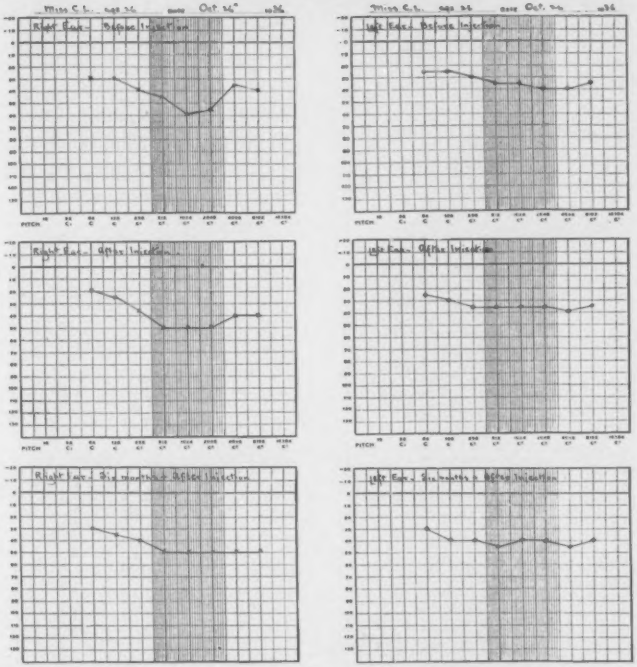
Duration and Character of Defective Hearing: Deafness of 3 yrs. duration. Buzzing in L. ear synchronous with pulse and of high pitch.

Contributory Factors: Frequent head colds since childhood.

Other Symptoms: Paracusis definitely present. Tinnitus: high pitched in L. ear only.

Eustachian Tubes: Both tubes partly patulous.

Drum Membrane: Slightly dry in surface, both R. and L. ears.



Notes on Injection: Anesthesia perfect. Injection at point of election. Slight exudate.

Notes on Reaction: No discomfort, except feeling of fulness after injections.

Tinnitus: Greatly diminished.

Hearing Improvement: From 5 to 10 dcb. after injections.

Duration of Improvement: 17 mos. after injections shows still further improvement in several tone frequencies.

Patient's Observation: Hearing much clearer, especially with several voices speaking simultaneously.

CASE 5.

Familial History: Mother and maternal aunt otosclerotic.

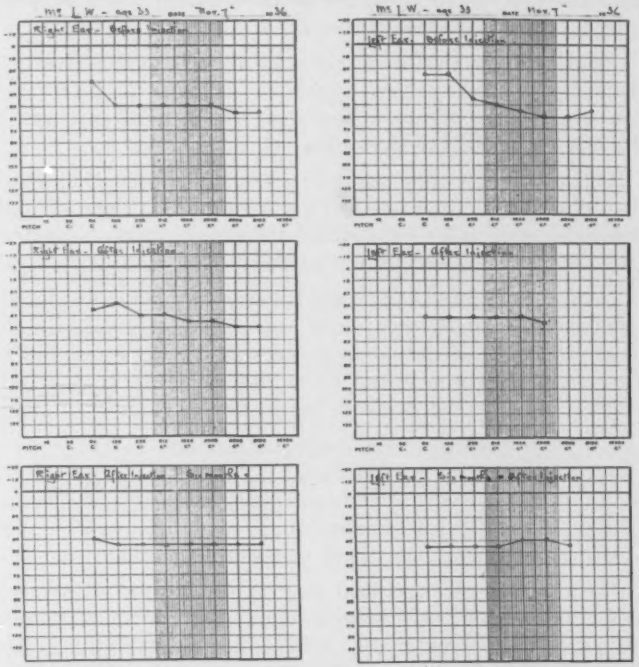
Duration and Character of Deafness: Five years, gradually increasing deafness.

Contributory Factors: Injury to R. ear drum about 10 years ago, punctured by penpoint.

Other Symptoms: Marked hyperthyroidism. Patient under active treatment. Marked continuous tinnitus; paracusis active.

Drum Membrane: Slightly retracted R. and L., but normal in color.

Eustachian Tubes: Both patulous.



Notes on Injection: Anesthesia perfect. Puncture at point of election. No exudate.

Notes on Reaction: No discomfort of any kind, except an itching in both ears, but this cleared up without difficulty.

Hearing Improvement: From 5 to 20 dcb. in some frequencies in both ears.

Tinnitus: Greatly reduced.

Duration of Improvement: 4 mos. after injections shows continued and increasing improvement.

Patient's Observation: Hears more distinctly and more accurately.

where the thyroxine fluid has gravitated. In but few cases has there been a slight fluid exudate about the site of injection; no pain, no feeling of discomfort and no vertigo has been noted. In no case has there been any inflammatory reaction following this treatment. The only sensation that patients complain of is a feeling of fulness in the ear.

Injections are undertaken in the office, preferably in the regulation treatment chair, in which a recumbent position may be secured, if necessary. Following the injection, the patient is instructed to remain quiet for 20 to 30 minutes and is further cautioned not to be too active for the rest of the day.

The thyroxine treatment consists of four consecutive injections made alternately in right and left ear at intervals of one week. This allows a period of 14 days in each ear for the absorption of the thyroxine solution.

Suffice it to say that the injection should be done under the best possible illumination of the membrana tympani, and I usually employ a lens for closer inspection of the membrana tympani and its detailed morphology.

Prior to beginning the thyroxine treatment, a careful audiogram should be made and a check-up or second audiogram may be of additional advantage in order to secure accuracy. In all of these cases I have used the 2-A audiometer, and this is tested to its full-registered capacity before the audiogram is begun.

A second audiogram is made within two to three weeks after the last (fourth) injection. To determine the stability of this therapy, when improvement has been noted in the second audiogram, a third audiogram is taken within six months to a year following this short course of treatment.

The selection of cases that may be best qualified to receive such treatment is based largely on our ability to make a diagnosis of clinical otosclerosis. Where there is a familial history of progressive deafness, audiometric and tuning-fork findings of defect in upper and lower tone limits, negative Rinné, patulous Eustachian tubes, fairly normal membrana tympanum, the presence of tinnitus, paracusis Willisi, and progressive decline of hearing in one or both ears — it is, in my opinion, a fairly stable evidence that we are not dealing with either nerve deafness *per se* nor a purely sound-conducting deafness.

One of the salient factors in the selection of cases for this treatment, as observed personally by Gray to me, was that

CASE 6.

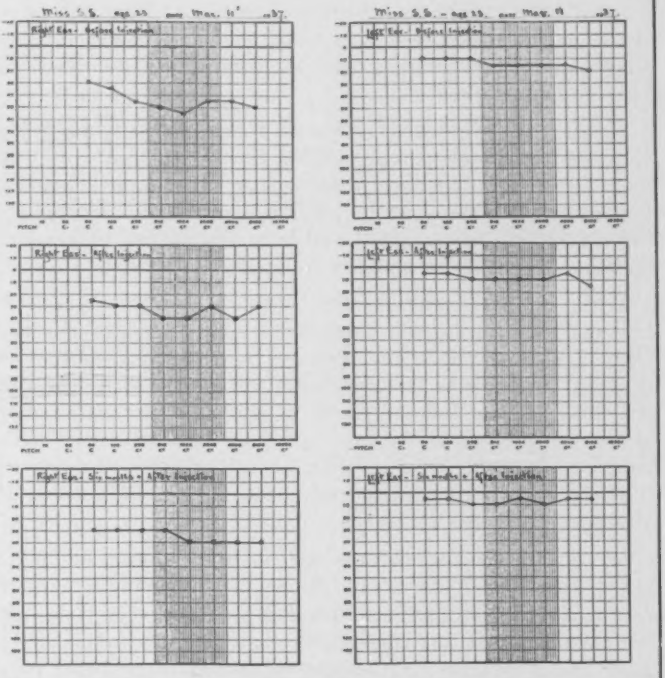
Familial History: No evidence of defective hearing in family other than brother now under similar treatment.

Duration and Character of Deafness: Several years high pitched tinnitus, worse in R. ear.

Other Symptoms: Mucosa of nose edematous. Huskiness of voice noticed for years.

Drum Membrane: Not particularly retracted; color good.

Eustachian Tubes: Both patulous.



Notes on Injection: Anesthesia perfect. Puncture at point of election. Slight exudative drip on first injection.

Notes on Reaction: No discomfort.

Hearing Improvement: From 5 to 20 dcb. in R. ear and 5 to 15 dcb. in L. ear.

Duration of Improvement:

Patient's Observation: Greater clarity in hearing voices. Better hearing at longer range.

patients over 30 years of age usually would be less satisfactory subjects for the thyroxine treatment, due either to their age or the long duration and progressive character of their defective hearing.

This series of 42 cases has been divided into three groups as far as results may be noted:

Group 1: No improvement following thyroxine therapy. In this group we must tabulate nine cases.

Group 2: Improvement noted in 10 cases of comparatively small decibel changes with the audiometer in less than two or three tone frequencies and in which there was some recession shown in the third audiogram taken within six months to one year following treatment.

Group 3: Includes 23 cases in which there were definite changes in the audiogram, ranging from 5 to 50 dcb. improvement in each ear in more than two or three tone frequencies throughout the tone scale and in which the accomplished improvement has remained stable at the taking of the third audiogram within six months following treatment.

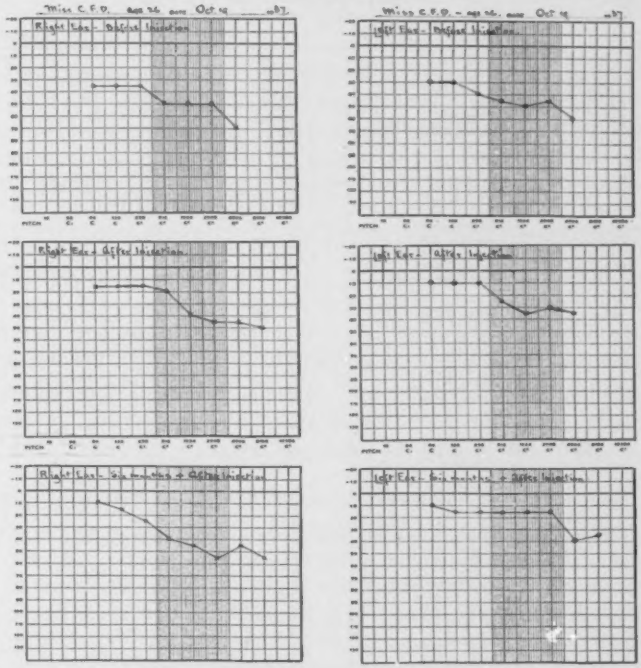
While the end-results of the thyroxine treatment have not reached quite as high a percentage level of improvement as Gray has indicated in his series of 14 cases, I would submit that our hearing tests have been more accurately recorded, for Gray did not use any form of audiometric measurement, and I have consistently and carefully applied every audiometric measurement in addition to tuning forks to determine the hearing status at the most susceptible intervals in the observation of this therapy.

My end-results, therefore, can be briefly reported as follows: Of the 42 cases subjected to thyroxine treatment, 23, or 54.8 per cent, have shown definite improvement in hearing capacity, and in some cases cessation or reduction of tinnitus and a general clarification in speech perception; 10, or 23.8 per cent, show slight improvement by comparison of audiograms before and after injection but this was not stabilized in the final audiograms six months+ later. In nine, or 21.4 per cent, no improvement was noted following treatment. In no case have I observed any decrease in hearing following these injections.

Even though this presentation cannot include all minute records of the 42 cases reported, I desire to add that most

CASE 7.

Familial History: No record of deafness for several generations.
Duration and Character of Deafness: About three years.
Other Symptoms: Tinnitus in both ears.
Drum Membrane: Normal.
Eustachian Tubes: Both patulous.



Notes on Injection: Anesthesia perfect. Injection at point of election. Slight exudate. Slight bulge in drum membrane, showing position of injected fluid.
Notes on Reaction: No discomfort except feeling of fulness after injection.
Hearing Improvement: From 5 to 50 dcb. after injections in L. ear and 15 to 30 dcb. in R. ear.
Tinnitus: Still present but somewhat diminished.
Duration of Improvement: 6 mos. after injection L. ear shows decided improvement throughout voice range and patient now hears 8,192 d.v. at 35.
Patient's Observation: Has clearer perception and less tinnitus.

CASE 8.

Familial History: No history of defective hearing.

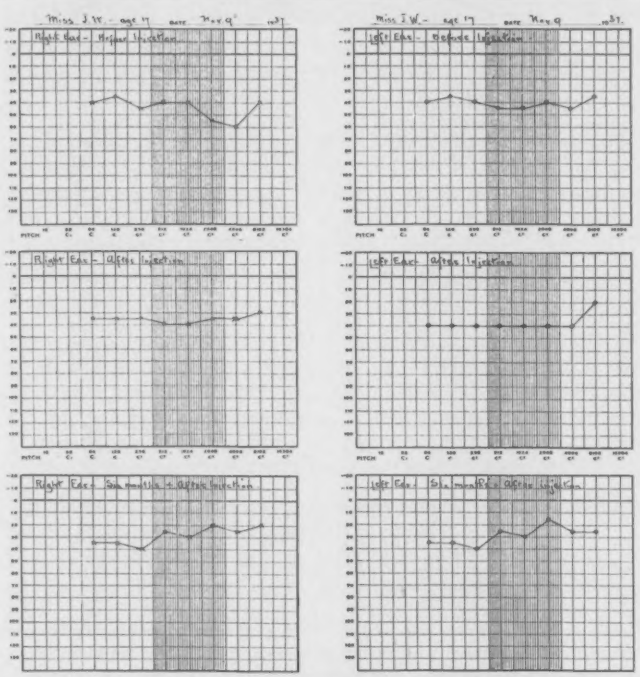
Duration and Character of Deafness: Actively noticeable during past year.

Contributory Factors: Blood serology, cytology and chemistry all within normal limits. Basal metabolism, minus 12.

Other Symptoms: Negative Rinné (both), moderate tinnitus (512 d.v.).

Drum Membrane: Unusually small external ear canal.

Eustachian Tubes: Both patulous.



Notes on Injection: Anesthesia perfect. Posterior quadrant and posterior lateral wall of external auditory canal in L. ear form a peculiar angle, projecting forward, and needle had to be advanced slightly to left toward manubrium. No exudate.

Notes on Reaction: No discomfort, except usual feeling of fulness.

Hearing Improvement: Marked improvement in both ears.

Duration of Improvement: Five months after injection further improvements registered in audiogram.

Patient's Observation: Clarity for all sounds.

careful observations have been made of each case, carefully tabulated, and they can be utilized for further study.

An interesting feature of the cases that show definite improvement is the fact that three of these were hyperthyroid cases with basal metabolism ranging from —five to —20.

Unfortunately, Gray, whose intensive study of otosclerosis for a period of 25 years and who originated this thyroxine therapy, passed away in January, 1936, in the midst of his research of this problem.

The question frequently presented is: "If improvement can be obtained in a certain percentage of cases subjected to this therapy in a first series of four injections, why would not a second series of injections at a later period be subject to the same therapeutic reaction, and why would it not be possible to obtain further improvement?"

Finally, I am mindful of the fact that this therapy is quite empirical in character and the explanations that might be made of the reason for such improvement are still somewhat vague, but the data are as accurate as can be obtained by modern methods of determining the acuity of hearing.

Following the publication of Gray's paper on Thyroxine Therapy in October, 1935,[4] and in my own preliminary report in February, 1936,[3] it is rather disappointing to me that more of our colleagues have not carefully considered this therapeutic approach in the treatment of otosclerosis, always, until now, a more or less hopeless outlook in our otological work. If the impetus given by this report will induce many of our colleagues to experiment further with this therapy and if the consensus of their reports can be gathered and studied, there may be a fruitful source of research and, possibly eventually, a fair percentage of otosclerotic cases showing improvement by this therapy.

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SYMPOSIUM.

WHAT IS JUSTIFIABLE TO DO IN OTITIC MENINGITIS?

(a)—A BRIEF (HISTORICAL) REVIEW OF THE OPERATIVE TREATMENT OF PURULENT MENINGITIS.*

DR. THOMAS J. HARRIS, New York.

By general consent, meningitis is considered the most formidable complication of diseases of the ear and, up to the present time, the most baffling to treat. Statistics show almost 100 per cent mortality in all forms of meningitis, formerly known as brain fever. Even those present today remember when a case of meningitis was regarded as hopeless, so far as treatment was concerned and, like a case of the plague of old, certain of but one outcome.

Until about 75 years ago, no attempt was made at surgical approach. It remained for Gross,[1] in 1873, to stress the necessity of surgical intervention and evacuation. A number of years elapsed, after Gross had proclaimed this truth, before any surgical measures were resorted to. The first surgical step was *ventricular puncture and drainage*. Von Bergman[2] was the first to perform this operation, in 1888. He was followed by von Beck,[3] who reported a case of acute hydrocephalus, following chronic otitis media where ventricular puncture was performed, with the cure of the case. He also reported, in 1896, a case of traumatic cerebral abscess followed by purulent meningitis, which recovered in 126 days after puncture of the lateral ventricle. Quincke,[4] in 1888, performed the same operation, and Robson,[5] in 1889, reported a case with recovery, by this method of treatment. This procedure, while encouraging, was far from giving the desired results, and the next year, 1890, Wynter[6] introduced the operation of *draining*, in the lumbar region, *the spinal subarachnoid space*, and a year later (1891) Quincke first operated by the method of lumbar puncture still in use at the present time. Shortly afterwards (1891) Ballance[7] was the first to operate by

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*Read as part of a Symposium, "What Is Justifiable to Do in Otitic Meningitis?" before the Seventy-first Annual Meeting of the American Otological Society, Inc., Atlantic City, May 5, 1938.

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the occipital route, and the same year Morton[8] demonstrated that the foramen of Magendie is never closed, showing that the posterior subarachnoid space is capable of being drained. Finally, in 1902, Rolgaus[9] attempted a third method of treatment, namely, the *introduction* of *drugs into the subarachnoid space*, but without success. About the same time, the Italian otologist, Gradenigo,[10] combined the operations of craniotomy and lumbar puncture in three cases of leptomeningitis of otitic origin, with recovery in all three. Hinsberg,[11] about 1904, reported seven cases of otitic meningitis cured and five temporarily improved by drainage of the subarachnoid cavity. Kümmel[12] reported, in 1905, a successful operation for purulent meningitis by double trephine of the occipital bone and the institution of adequate drainage. The reports obtained from this operative procedure were encouraging, but it remained for Haynes,[13] in 1913, exhaustively to cover the subject in a paper read before the American Laryngological, Rhinological and Otological Society, entitled, "Surgical Treatment of Meningitis." In this paper, Haynes described an operation for draining the cisterna magna and set forth its advantages. While the desired results did not follow its employment, the paper of Haynes served to arouse widespread interest in the subject. In 1913, Day[14] published a report of 53 cases of otitic meningitis operated upon by various methods, with autopsy reports in 38. The surgical measures employed included dural incisions, drainage of the lateral ventricles, through-and-through irrigation from the lateral to a needle in the lumbar canal, and drainage of the cisterna magna. Day's conclusions were that no improvement in the mortality rate was noted. Eagleton,[15] one of our most indefatigable fellow members, in his attempts at a successful solution of the problem, in a series of papers from 1921 to 1924, reported his results by various methods: subarachnoid irrigation from a point over the frontal lobe to the cisterna magna; subarachnoid irrigation with replacement of fluid withdrawn with Ringer's fluid; ligation of the homologous common carotid artery. In 1932, at the annual meeting of this Society, Kerrison[16] presented a carefully prepared paper on the "Treatment of Otitic Leptomeningitis with a Plea for Investigation in Two Directions, Kubie's[17] Theory of Forced Drainage; and Direct Surgical Drainage." Kerrison in the paper recommended the method of Kubie of forced drainage. This operation was set forth by Kubie two years later, in 1934, in a

paper read before this Society in collaboration with Zabriskie and Hare, entitled, "Forced Drainage for the Treatment of Meningitis Secondary to Ear and Sinus Infections." This procedure has been tried by numerous operators, but in spite of the promise offered, the operation has not given the desired results. No new surgical procedures were presented after this until prontylin, in conjunction with prontosil administered intravenously or intramuscularly, was used on the Continent and in England, and more recently in this country with the remarkable results to be reported by Dr. Applebaum, covering a long series of cases of septic meningitis, especially of streptococcic hemolyticus origin.

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SYMPOSIUM.

WHAT IS JUSTIFIABLE TO DO IN OTITIC MENINGITIS?

(b)—SURGICAL INDICATIONS IN SUPPURATIVE MENINGITIS.*

DR. JAS. G. DWYER, New York.

Broadly speaking, we may say that the surgery of meningitis has as its primary object the removal of the focus of infection — in other words, the surgery of meningitis is the surgery of the focus. This is not as simple as it may seem, and a thorough study of each case must be made before we can establish the site of the focus — if, indeed, this is possible in a given case. We must have some clue to the pathway of infection and a thorough knowledge of the pathology and bacteriology. Again, we must emphasize the fact that all cases of meningitis are not surgical ones. In some cases the surgery is all-important; in others, surgery is useless, if not contraindicated. This point will be emphasized later when dealing with the kinds of meningitis and the pathways of infection. Hence, a broad comprehensive and all-embracing picture of meningitis must be constantly kept in mind in dealing with an individual case and any little lead or indication may be of the utmost importance in influencing the handling of such a case.

The primary requisite in meningitis, as in other diseases, is the proper diagnosis. This cannot be emphasized too strongly. Hence, we have to ask ourselves at the outset two questions: *1.* Is meningitis present? *2.* If present, what kind is it? The exact diagnosis of meningitis is made essentially from an examination of the spinal fluid. The clinical symptoms are familiar to everyone, but it cannot be emphasized too strongly that the final diagnosis depends upon our findings in the spinal fluid and our interpretations thereof. Another important clinical pathological aid is the blood examination, which will be dealt with later.

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EXAMINATION OF SPINAL FLUID.

Great emphasis must be placed upon our findings in the spinal fluid because the surgery to be employed rests, in the last analysis, upon the results of this examination. In going over the literature of the last few years on meningitis, we can draw no conclusions as to the mortality rate, because the reports are faulty in some respects, are incomplete in others and no conclusions can be drawn. This applies especially to the reports on the spinal fluid; moreover, meningitis is rather rarely seen by any one surgeon, outside of the larger centres, and it is essential to understand and analyze what the spinal fluid does or does not tell. This is so important that we advocate that part of the training of the otologist should include an actual working knowledge of the examination of the spinal fluid.

The following is the graphic picture of what we should look for in spinal fluid analysis and the generally accepted interpretation of the findings: The fluid is examined under five different headings, classified as follows: *1.* Physical; *2.* Chemical; *3.* Cytological; *4.* Serological; *5.* Bacteriological. These examinations will be dealt with under each classification, together with the deductions to be drawn and their limitations. Again, we emphasize that these examinations under the various headings dovetail with each other.

In order to take advantage of every clue in the examination of the spinal fluid, it is essential to carry out a certain routine in collecting the specimen. For some of the tests, especially chemical, it is important that the fluid should be examined as soon as possible. One should have handy, in addition to receptacles for taking the fluid, an alcohol lamp and a test tube of paraffin with a very low melting point. Just before the spinal puncture, this paraffin should be melted. Immediately after the withdrawal of the fluid, it should be covered with a film of this melted paraffin to seal it. It is well to divide this specimen into two parts, sealing both. If the comparative blood examinations are made at the same time, the blood should also be sealed. It is extremely difficult to get the proper interpretation of spinal fluid findings from the ordinary textbook on clinical pathology, and hence this work is here dealt upon *in extenso*.

1.—PHYSICAL EXAMINATION.

Normal spinal fluid is colorless, like water, and usually comes out either drop by drop or at least without much force. The fluid ordinarily remains clear upon standing, but sometimes a slight pellicle may be seen in some specimens; however, this seldom occurs.

Pathological spinal fluid may or may not be cloudy. This cloudiness varies from a very slight, almost imperceptible one to a dense, thick, opalescent one. If blood is present in the fluid or if a blood vessel is punctured in performing the tap, the fluid may be blood-stained.

It is emphasized here that pathological spinal fluid, from the physical examination, may be clear or cloudy. If it is persistently clear, in the presence of the usual clinical signs of meningitis, we may be dealing with either a tubercular or syphilitic meningitis. The differential diagnosis in these types will be dealt with later. If the spinal fluid is cloudy, we know we are dealing with a pathological process, but we have to differentiate a cloudy fluid with organisms present and a cloudy fluid without organisms. This is an extremely important thing from the practical standpoint and from the standpoint of surgery.

A cloudy spinal fluid may be looked upon as the result of a tissue reaction — an exudation of cells, due to irritation of the meninges or cerebral substance or, in general terms, the cerebrospinal axis. This tissue reaction may be evoked by any sort of irritant, including an infection. If the infection, as it is this we are dealing with, remains external to the meninges, so that it does not penetrate the meninges, we get a cloudy fluid but no organisms in the fluid. If the infection has broken through, the spinal fluid is cloudy and contains organisms, and we have meningitis — usually diffuse suppurative meningitis. The exactly analogous situation is seen very often in the eye and the differential point stressed above has been recognized for years. Thus, analyzing the eye condition, we may have a pneumococcus or streptococcus ulcer of the cornea with the anterior chamber full of pus or hypopyon, and yet it has been recognized for years that unless the organism penetrates the cornea and invades the anterior chamber, the exudate or hypopyon is sterile — that is, free of organisms. In other words, the organisms are in the cornea, have not broken through

the basement membrane, but the tissue reaction sets up results in an exudate of leukocytes into the anterior chamber. This illustration from eye pathology thus applies to the pathology of meningitis. The organisms, by their action, irritate the meninges extradurally, cause an exudation of leukocytes and thus lead to a cloudy spinal fluid, but still free from organisms. On the other hand, if the organisms break through, we have a real general meningitis. Hence, the recognition that persistently cloudy fluids without organisms means an extradural infection and a strong indication to resort to surgery to remove the focus. It is in this type of case that the results have been so satisfactory. In the last five years, we have saved such cases by the recognition of the above fact. In such cases, a sequestrum of bone has been found and, upon its removal, the spinal fluid becomes clear and the symptoms of meningitis promptly disappear. Thus, the recognition of this point in the examination of spinal fluid is extremely important.

2.—CHEMICAL EXAMINATION.

During recent years, much work has been done on the chemistry of the spinal fluid, but not enough has been done to draw any conclusions as to the practical value of the various chemical tests. The relationship between the alkalinity of the blood and the spinal fluid, the various tests for choline and neutral red, etc., as outlined in the questionnaire, should be carried out in every case and the results compared.

From the chemical standpoint, normal fluid presents certain characteristics. Usually there is a copper reducing substance present, and it also has a trace of lactic acid and a smaller amount of globulin. Kopetzky has done a large amount of work on the spinal fluid, and reference to his work is made here for details. Chemically, pathological fluid shows an increase in the amount of lactic acid and globulin, and often the sugar reducing substance disappears upon the establishment of an acute diffuse meningitis.

3.—CYTOLOGICAL EXAMINATION.

Normal spinal fluid shows usually 3-5 cells per cmm. These cells are most often lymphocytes. Pathological fluid shows from a few cells up to thousands per cmm. From the cytological standpoint, we can evolve the following working rule: Given

the clinical symptoms of meningitis, the cell count can be interpreted as follows: If the spinal fluid is clear physically, and we have an increase in the cell count, especially in the percentage of lymphocytes, we are probably dealing with a tuberculous or syphilitic meningitis. If the former, we can sometimes demonstrate the tubercle bacillus directly by centrifuging the specimen for hours. If we cannot demonstrate it directly, we can inject a guinea pig with the fluid or inoculate Petroff's Media. If syphilitic, our serological examination will confirm the diagnosis by the Wassermann or Kahn test. If the spinal fluid shows leukocytes of the polynuclear or large mononuclear type, we next determine if bacteria are present. If bacteria are not present and do not appear, we are probably dealing with an extradural infection, as discussed above under cloudy fluids. If bacteria are present we are likely dealing with a diffuse general meningitis or true meningitis. The term likely is used, as we can conceive that a few organisms may spill over into the spinal fluid and upon the removal surgically of the focus, the spinal fluid becomes clear.

To recapitulate, a clear fluid physically, with an increased cell count, especially lymphocytes, is likely tubercular or syphilitic. A cloudy fluid physically with an increased cell count and no organisms may be an extradural infection and be amenable surgically to treatment. A cloudy fluid physically with an increased cell count and organisms usually means meningitis—usually of the diffuse variety.

4.—SEROLOGICAL EXAMINATION.

This has not much practical bearing in acute meningitis outside of the meningococcus type, because the serological reactions of the streptococcus has not yet been worked out; however, the organism found should be tried out against the antistreptococcus sera, and the pneumococcus could be classified in the usual way. Reference has already been made to the Wassermann and Kahn reactions.

5.—THE BACTERIOLOGICAL EXAMINATION.

This is, in some ways, the most important examination of the spinal fluid, because apparently the greatest errors in the diagnosis are made in this province. Thus, in the literature we read of cured cases of meningitis in which no organism was found in the spinal fluid; again, in some cases, the

organism was found in smear but not in culture. Again a mixture of several organisms were found. All of these points can be easily explained from the bacteriological standpoint. The organisms usually found are the meningococcus, streptococcus, pneumococcus and occasionally the staphylococcus, b. influenza, b. typhosus, etc. We are not concerned here with the meningococcus, as it is not a surgical problem, but we must be sure to exclude the presence of the meningococcus in an otitis case. We must be absolutely sure that the case in hand is one due to invasion of the meninges from the ear, nose or throat, and not a concomitant meningococcus infection. To do this we must keep in mind several bacteriological facts. The meningococcus, streptococcus, pneumococcus and staphylococcus are all cocci, and morphologically, at least in smear, can be mistaken for each other. However, the meningococcus is Gram— and all the others are Gram+. This is a differential test by smear. It must be borne in mind, however, that sometimes a Gram+ coccus becomes Gram— when undergoing degeneration and, hence, we have what appears Gram+ and Gram— in the same slide. In such a case, we are undoubtedly dealing, not with two organisms, but with the pneumococcus, streptococcus or staphylococcus and its degenerated forms and not a mixture of these with the meningococcus. This can be proven out by culture and serological reactions. The discrepancies between the appearance of an organism in smear and not in culture is easily explainable from the bacteriological standpoint. If the organism is dead or dying, due to the action of the normal bactericidal power of spinal fluid, we may have only a few virile organisms present and if we plant only a loopful, we may fail to get a growth. If we plant 1-2 cc. of the centrifuged specimen, we will get growth. This has happened to the writer time and again.

EXAMINATION OF THE BLOOD.

The blood count is often of help in the diagnosis of septic meningitis. In an adult, we usually find the blood count very high and also the polynuclear count high. The only other condition in which we find such a high blood count is from respiratory diseases, as pneumonia. Hence, if we are dealing with an adult and the blood count is high and the chest is clear, we are probably dealing with an infection of the meninges or central tissue. The writer has seen as high as 48,000

and a polynuclear count of 98 per cent. In the syphilitic and tubercular forms, we may have even a leukopenia with a relative lymphocytosis.

TYPES OF MENINGITIS.

From the surgical standpoint, the type of meningitis is important. We may, for purposes of discussion, divide the types met with into three classes: *1.* acute, *2.* subacute, *3.* chronic. By acute we mean the very fulminating type, such as that following dislocation of the stapes or direct injury to the meninges, or that in which the meninges are invaded suddenly and overwhelmed without any wall of protection being set up. These cases often come to a fatal issue in 48 to 72 hours. Surgery will probably not be of avail in these forms. The chronic are chronic relatively only, and by this we mean the type of case that runs a low course, lasts for five to six weeks and then succumbs. The vast majority of cases that we see lie in between the fulminating and the chronic and last a week to 10 days, and it is in this type that we have to use every element of use in making our diagnosis and handling the case properly. One type of meningitis that has to be emphasized that is commonly met with is the so-called ambulatory meningitis, in which the only clinical symptoms present are headache and a slight temperature. These types run a very low course until they suddenly go to pieces, sometimes weeks after a mastoid operation. This type of meningitis is most often due to the No. 3 pneumococcus.

PATHOLOGY AND PATHWAYS OF INFECTION.

The pathology is important in surgical meningitis. The primary reaction to an infection in meningitis is a protective one — in which we have a protective exudate formed almost gelatinous in character and this is looked upon as a protective mechanism, as the infection progresses. We have an extension in the form of an osteitis periostitis or osteomyelitis, and the pathology is that of a thrombophlebitis. We also get infection by direct extension, as in injuries to the dura, direct extension at the site of operation, etc. Meningitis may also occur in the course of a blood stream infection. The thrombophlebitis process can be easily demonstrated under the microscope. The infection follows probably most often the path of least resistance, through the cells of the mastoid for

instance, but we are coming to realize more and more that the pathway of infection is very important in every case, and most important in estimating any given case of meningitis from the surgical standpoint. The pathway of infection may be physical, as by direct extension from the nasopharynx through the Eustachian tube, then the mastoid and the meninges. We believe, however, with better analyses of the cases, that the pathway is often from the nasal accessory sinuses or the lymphoid tissue around the nasopharynx and that the pathology involved is that of an osteomyelitis or osteitis through the above described thrombophlebitic process. Again, we must realize that from the character of the bones of the skull, and especially some areas in the temporal bone, that we can get a true osteomyelitis, comparable to that in the long bones, due to the medullary matter present. Hence, our conceptions of osteomyelitis in comparatively small areas around the base of the skull, as compared with the larger areas in the long bone. These observations throw new light upon the pathology of meningitis, and show us how careful we must be in the diagnosis of the pathway, where this is possible. The recognition of this point is also of importance in our conceptions of extension of infection to the petrous portion of the temporal base. Some believe that we can only get infection through the cells of a pneumatic bone and that there must be a direct pathway through these cells to the petrous apex. Others believe that the infection can be carried along the blood vessels and appear at a distant point and that no direct physical pathway exists and that, for instance, an otological infection may supervene from a sphenoid infection by infected blood vessels in the form of a thrombophlebitic process.

CHART OF SPINAL FLUID FINDINGS.

Below is a graphic analysis of the main points in the diagnosis of meningitis from the cerebrospinal fluid. In a suspected case of meningitis — that is, one clinically diagnosed as a suppurative meningitis — the following outline will be of advantage in the interpretation of the spinal fluid findings:

1.–PHYSICAL.

a. Spinal fluid persistently clear, look for tubercular or syphilitic meningitis.

b. Spinal fluid persistently cloudy without the presence of organisms — an extradural lesion and exploratory operation is indicated.

c. Spinal fluid persistently cloudy with organisms — usually suppurative meningitis.

2.–CYTOLOGICAL.

a. Clear spinal fluid with an increase in lymphocytes — tubercular or syphilitic meningitis.

b. Cloudy fluid with an increase mainly in the polynuclear leukocytes and no organisms — an extradural lesion as above.

c. Cloudy fluid with an increase mainly in the polynuclear leukocytes with organisms — suppurative meningitis.

3.–BACTERIOLOGICAL.

a. Gram positive cocci present in smear or culture or both — streptococcus, pneumococcus or staphylococcus meningitis. The staphylococcus is rare in rhino-otological work — identify the organism further as usual.

b. Gram negative coccus. This is meningococcus or the epidermic form of meningitis.

c. Both Gram positive and Gram negative cocci present probably streptococcus or pneumococcus meningitis with degeneration forms — identify further as usual.

Subjoined is the questionnaire used by the New York Committee of the American Otological Society, Inc. It is comprehensive and outlines all the data necessary in a case.

REPORT ON CASE OF MENINGITIS.

1. By whom report is made.
   Name .....
   Address .....
2. Institution in which case was treated.
3. Previous ear, nose and throat history.
   (a) Otitic infections:
      1. How many? .....
      2. When was last one? .....

(b) Paranasal sinus infections:

1. How many? .....
2. When was last attack?.....

4. Clinical history of case.

(a) Give details as to onset of earache:

1. Did it come on before paracentesis?.....
2. Was there an IMMEDIATE flow of pus?.....
3. Did earache persist AFTER paracentesis?.....
4. Was secondary paracentesis necessary?.....

5. Neurological findings at beginning and during course.

(a) When were first neurologic signs noted? What were they?.....

1. Headache — where localized — character.....
2. Nystagmus — direction — character.....
3. Babinski — strong — weak — time of onset.....
4. Kernig — strong — weak — time of onset.....
5. Diplopia — external rectus paralysis — time of onset.....
6. Dermography .....
7. Hyperesthesia .....
8. Sensorium — time of clouding — general characteristics.....
9. Opisthotonos — time of onset — strong — weak.....
10. Vomiting — time of onset — characteristics.....
11. Temperature — high — moderate — low — type of temperature curve:
    (a) Septic type .....
    (b) High level .....
    (c) Low level .....

6. Ophthalmic findings — eye grounds, fields, etc.

7. Blood examinations — hemograms.

8. Spinal fluid findings*

(a) Cell count, and types of cells found.....

(b) Bacterial examination .....

1. On smear .....
2. On culture .....
    (a) Aerobically .....
    (b) Anaerobically .....

(c) Chemical examination.

1. Albumin .....
2. Globulin .....
3. Copper reduction substance.....
4. Lactic acid content in relation to lactic acid in patient's blood plasma .....
5. Ph. content in relation to Ph. in patient's blood plasma.....
6. Chloride content in relation to chloride in patient's blood plasma .....
7. Carbonate content in relation to carbonate in patient's blood plasma .....
8. CO_2 content in relation to CO_2 in patient's blood plasma.....
9. Choline .....

(These chemical examinations should be taken every 2-3 days during the course of the disease and recorded serially.)

(d) Physical characteristics of the fluid.

1. Specific gravity .....
2. Color .....
3. Translucency .....
4. Pedicle — character and shape.....

*All spinal fluid examinations should be made immediately after fluid is withdrawn.

9. Procedures as to treatment, both medical and surgical.
   (a) Procedure as to treatment.
      1. When was treatment started?.....
      2. What was its nature?.....
   (b) Describe surgical procedure.

10. Consultation and opinions.
   (a) Give interval between onset of otitic lesion and mastoidectomy.....
   (b) Is meningeal infection result of acute or chronic ear lesion?.....

11. Outcome of case.
   (a) Final physical findings.....
   (b) Autopsy .....
      1. General .....
      Microscopic .....

12. Filing of specimen from operation or autopsy, etc.

13. Was case seen by Committee?.....
   (a) By whom? .....
   (b) Opinion .....

14. Remarks by Committee .....
.....
.....
.....

300 Park Avenue.

SYMPOSIUM.

WHAT IS JUSTIFIABLE TO DO IN OTITIC MENINGITIS?

(c)—THE SURGICAL TREATMENT OF MENINGITIS.*

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Any discussion of the surgical treatment of meningitis entails first, the consideration of certain basic facts concerning the meningeal infection. Meningitis has been divided into various forms based upon the cytologic, chemical and bacteriologic examinations of the cerebrospinal fluid and if these criteria of differentiation are accepted, one might conclude that infection occurs in the cerebrospinal fluid rather than in the tissues which comprise the meninges, a conclusion which is obviously erroneous. It has been our belief that the various forms of meningitis as differentiated by laboratory examinations, particularly in the presence of similar clinical phenomena, are all due to an infectious process within the meninges and all the differences are merely of degree, extent, and perhaps type of infection. Despite much that has been said to the contrary, this still seems to us the most logical viewpoint.

It follows, therefore, that any operation designed for the cure of meningitis, which has as its sole purpose the drainage of the cerebrospinal fluid, whether that drainage be established through the basal cisternae, as described by Horsley,[1] Ballance,[2] Parkin,[3] Murphy,[4] Haynes,[5] Day,[6] Haynes,[7] Dench,[8] Milligan,[9] Kümmell,[10] Kopetzky,[11] Kerrison,[12] Dandy[25] and others, or by laminectomy as advocated by MacEwen,[13] Göbell,[14] Barth,[15], Leighton and Pringle,[16] Rolleston and Allingham,[17] Rainey and Alford,[18] Spurling[19] and others, or by repeated lumbar punctures (MacEwen,[13] Kümmell,[10] Netter,[20], S. MacEwen Smith,[21] Tedeske,[22] Lamar,[23] Royster[24] and Dandy[25]) must of necessity fail of its objective.

From anatomic studies Davis[26] found that pus from the labyrinth first entered the lateral prolongation of the cisterna

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pontis, then extended into the cistern, spreading up and around the cerebral cortex. The theory that localization of infection takes place in the lateral prolongations of the cisterna magna and cisterna pontis, and that drainage of these areas through a dural defect, Jenkins,[27] Dench,[8] contributes to a cure of these cases, is in our opinion untenable because we have seen the walls of the basal cisternae covered thickly with streptococcal exudate despite the institution of continuous drainage. We do not mean to imply that the extension of infection in the meninges takes place solely by diffusion in the cerebrospinal fluid. In all probability meningeal infection is spread by contiguity and through the vessels of the pia. The value of drainage of the cerebrospinal fluid is purely theoretical and is based on the idea that the relief of pressure increases the local circulation, bringing more blood and more leukocytes to the area, thus enhancing the local resistance.

In 1921, Eagleton,[28] in one of his comprehensive papers on the treatment of otitic meningitis, made the statement that "suppurative meningitis should be regarded not as a single disease, but as a group of diseases of the cerebrospinal system." This is another of the basic truths that must be grasped before one can develop a rational surgical approach for the treatment of this condition. There are cases of meningitis due to various bacteriologic agents associated with an otitis in which the latter is an incident in the disease picture rather than an etiologic factor. Meningococcus meningitis is an example. There are, in addition, some cases of pneumococcic meningitis which develop an otitis and a meningitis a few hours after the beginning of an acute upper respiratory infection. Often the spinal fluid in these instances looks like an emulsion of organisms and it contains comparatively few cellular elements. The ear cannot justly be regarded as the focus in these instances and we believe any surgical approach, so far as the temporal bone is concerned, offers little possibility of relief.

Otitic meningitis has been divided by Eagleton[28] into three groups: *1.* Cases secondary to sinus thrombophlebitis; *2.* middle fossa cases in which the infection extends through the tegmen tympani, and *3.* the labyrinth cases. Politzer[29] classified meningitis as: *1.* Pachymeningitis externa circumscripta (extradural abscess); *2.* pachymeningitis interna circum-

scripta (intradural abscess), and *3.* leptomeningitis diffusa and meningitis serosa. There are many variations of these classifications in the literature.

The point of view advanced by Eagleton in 1921, and accepted by many otologists, that only those cases should be classified as healed suppurative meningitis, in which organisms had been demonstrated in the spinal fluid, is at variance with the opinions of Lund[30] and others. Lund regards the presence of few cells in the spinal fluid as abnormal and an increase in the cell count of more than 5 cells per cm. significant of meningitis. These apparently extreme differences of opinion as to what constitutes "meningitis" are possible of understanding provided we regard them from the point of view of operative interference and not diagnosis. The diagnosis based on the spinal puncture findings as advocated by Lund has been utilized as an indication for therapy and he believes that it has materially cut down the mortality statistics in otitic meningitis. The criticism of this statement is the usual one, namely that many of the cases did not have organisms in the spinal fluid, and therefore they were not true bacterial meningitis.

It seems evident that some change in our attitude must obtain in order that the therapy of meningitis and its indications be clarified. The changes in the spinal fluid, whether there be only an increase in the cell content or presence of viable micro-organisms, are to our mind, expressions merely of the degree, severity and extent of the inflammation in the meninges. These may result from an otitic focus wherever this focus may be. Delaying operative intervention in order to await bacteriologic culture reports will, according to Neuman,[31] occasionally sacrifice that little chance for recovery which a very sick patient may have. The duration of the acute meningitis is an important prognostic factor. Dench,[32] Mygind[33] and others have said substantially the same thing, namely, the longer the duration of the meningitis, the more grave the prognosis. Mygind, in discussing otitic meningitis, declared, "other surgeons may, like me, have operated on a number of consecutive cases of short duration without being able to avert a fatal issue." Nevertheless, he feels that operation should be performed without any waste of time. He further states, "that in a certain number of cases of meningitis, the inflammation of the brain does not set in as

in the majority of cases, with a sudden explosion like that of a gunpowder barrel into which a spark has fallen, but resembles rather a smoldering fire spreading for a short time to all sides and bursting at last into an all-destroying conflagration. In such cases, if the primary focus is eliminated early, the fire dies out for want of fuel." In such cases early and repeated lumbar puncture may show significant changes.

It has been stated by Lermoyes,[34] Dench,[35] MacEwen Smith[21] and others that in their experience the labyrinth was the most common pathway of extension to the meninges. Turner and Reynolds[36] believe that their observations seemed to indicate that the tegmen tympani was the most common pathway of the infection.

According to our own histopathologic findings and our clinical experience, we have found petrous pyramid suppuration to be the most common pathway of extension to the meninges. Obviously, these differences of opinion must be based to a considerable extent upon differences in the type of clinical material handled by the various reporters. Our material is composed predominantly of acute cases, whereas other clinics may have a preponderance of chronic middle ear suppurations with their resultant complications.

For the purposes of discussion of the surgical therapy of meningitis, we have divided meningitis into that secondary to: *a.* the labyrinth; *b.* sinus thrombosis and thrombophlebitis, and *c.* infection in the middle ear, mastoid and petrous pyramid. This is essentially the classification advanced by Eagleton years ago. We have purposely excluded meningitis due to intramedullary suppuration.

I. Meningitis Secondary to Infection in the Labyrinth.

In the instances in which the meningitis is secondary to a suppurative labyrinthitis, the tendency for the infection to remain localized and heal is exceedingly slight and the indications are for prompt labyrinthectomy, with inspection of the dura of the middle and posterior fossae.

There is a much higher incidence of mortality from the meningitis resulting from labyrinthitis secondary to an acute otitis media, or stapes dislocation, than from cases of labyrinthitis secondary to chronic suppuration of the middle ear. We have used as indications, while observing these patients,

fever, headache or any clinical evidences of an extension to the meninges. Repeated examinations of the cerebrospinal fluid are of great value.

II. Meningitis Secondary to Infection in the Lateral Sinus.

In sinus thrombosis or phlebitis, meningitis occasionally develops and every effort is made to handle the infection by getting well beyond the disease limits, proximally and distally laying the sinus open widely. If the bulb is thrombosed and purulent thrombi are present, we endeavor to secure drainage from the bulb by irrigating it with Dakin's Solution. We have sometimes introduced a soft catheter through the horizontal portion of the sigmoid sinus into the bulb.

III. Meningitis Secondary to Infection in the Middle Ear, Mastoid and Petrous Bones.

Needless to state, in all these cases a complete mastoidectomy is first performed. If the mastoid has been previously operated on, a careful systematic search is made for leads into the petrous pyramid, the peribulbar cells, the zygoma, exposing the superior and posterior semicircular canals. We employ the Eagleton[37] type of exploration. The exploration is not considered complete until every possible pathway of extension has been carefully investigated. We are not convinced of the utility of opening the dura in the treatment of meningitis as advised by some otologists.

CONCLUSIONS.

1. Meningitis is an infection of the tissues of the meninges, not the cerebrospinal fluid.

2. Drainage of the cerebrospinal fluid while it is useful is inadequate so far as cure is concerned.

3. While it is true that meningitis should be regarded as a group of diseases, the differences are chiefly: *a.* in the type of infections present; *b.* the pathway of the infection, and *c.* the tempo in which the infection develops.

4. Meningitis is usually associated with significant changes in the cerebrospinal fluid as obtained by lumbar puncture.

5. **As soon as these changes are discovered, the indication for the eradication of the suppurative focus is established. In the last analysis, surgery of meningitis is the early and adequate drainage of the focus from which the meningitis takes its origin.**

6. **This is all the more important now, since in sulfanilamide we have an adjuvant, that, provided the focus which feeds the meningitis be eradicated, will help us cure many cases of this hitherto almost uniformly fatal condition.**

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DISCUSSION.

Dr. Wells P. Eagleton (Newark): Dr. Friesner has covered the subject so well that very little remains to be said; and still one has to review in his mind, how one has changed; that is, I want to review how I have changed.

First, the removal of the focus of infection is still fundamental. Now that we have a drug that seems to be almost miraculous in its action, we should remember that there is still a focus, that is the cause of the meningitis, and unless we remove it, the case is apt to ultimately die.

The most important factors favoring recovery in meningitis, with bacteria in the fluid, lie in (a) the fixation of the infection to a localized area in the bone, and (b) the presence of a "protective meningitis."

With (a) eradication of the bony, or vascular focus, and (b) drainage of the infected collection of fluid, lying adjacent to it, (c) before the infection has become generalized in the leptomeninges, many patients should be cured, especially now with the aid of the salts of sulfanilamide.

I collected and analyzed for the New York Academy of Medicine, at their invitation in January of this year, all the cases (178) of meningitis in which there were germs in the cerebral spinal fluid, reported in the literature that have recovered.

Protective Meningitis: In the cases of meningitis with a high cell count, as Dr. Dwyer mentioned, with few or no bacteria in the fluid, the mortality should not be higher than 5 per cent, if the primary focus is promptly removed.

Localized Bacterial Meningitis: In the previous paper, which has not yet appeared, I showed where these separate bony foci are situated, and the districts of the arachnoid mesh that are first invaded. The symptoms from the different areas are very definite and distinct.

Primary Foci of Bony Infection and Areas of Meningeal Suppuration: The anatomical position, and order of frequency, according to my experience, where these bony foci and adjacent arachnoid areas are localized are:

1. Meningitis from the angle of the petrosal (where several plexii of veins goes outward into the sinus and meninges). (Eagleton, W. P.: Routes of Venous Vulnerability: Cerebrospinal Fluid. *Jour. Laryngol. and Otol.*, p. 728, Nov., 1929.)

In these cases there is usually a microscopic caries of the cancellous tissue which constitutes the angle, the lateral arm of the basal cistern being filled with infected fluid.

2. Infection in and around the saccus endolymphaticus, with a thrombophlebitis of the small vein in the sacculus, which results either from (a) an

osteomyelitis of the adjacent cancellous tissue or by way of (b) a retrograde thrombophlebitis ascending from the jugular bulb.

If the infected vein breaks down, an empyema of the saccus endolymphaticus is formed. Clinically there is no chill in cases of osteomyelitis in the saccus cases as in those of thrombophlebitis.

Saccus empyema is usually associated with a deaf labyrinth, but not always. If an unlocking operation, fully exposing the sacculus region, is performed before general invasive leptomeningitis has developed, most patients with meningitis from saccus infection recover.

3. Meningitis within the internal auditory meatus following infection of the membranous labyrinth. The meningitis is at first localized in the arachnoid prolongation surrounding the meatus.

Our procedure is to drain the labyrinth through the middle ear and then to evacuate the collection of fluid within the internal auditory meatus by incision of the dura over the prolongation.

4. Old suppuration in the labyrinth, resulting in late meningitis. In all cases of meningitis that present posterior fossa symptoms—stiff neck and reduction of the induced nystagmus to turning with hearing—"unlocking of the posterior half of the pyramid" is our first procedure.

Freeing and retraction of the lateral sinus allow a complete exposure along the posterior surface of the petrous pyramid as far as the adhesion of the dura to the outer lip of the internal auditory meatus. The saccus is either cut or torn through in the approach to the meatus. If properly performed the operation does not occasion a cerebrospinal leak nor cause deafness.

5. Meningitis from infection of the petrous apex. Fifty per cent of all intracranial complications that result from aural suppuration come from infection in the apex.

In all cases of otitic meningitis without a demonstrable point of entrance of the infection, I now "fully unlock" and thus expose the apex before opening the dura.

6. Jugular bulb cases, with infection of fluid in the cistern of the lateral inferior cerebellar recess. In these cases, in addition to the simple evacuation of the infected fluid, we must eradicate the surrounding area of infected medullary bone—the localized osteomyelitis. This is necessary because if this area is not removed the infected small veins continue to supply infection into the meningeal mesh.

Principles of Treatment: In all of the above six groups of cases, if you remove the focus of infection when there is (a) localization of the suppuration in the bone, but (b) before the onset of general meningitis, you will cure your case. If, however, you wait or perform a simple mastoid, the patient is apt to die; although today, with prontosil and prontylin, such mortality has been tremendously reduced.

You must remove the primary focus of infection in the bone or vessels, for if this is not done, even though you give them sulfanilamide, a large percentage of them will relapse. These cases are deceptive because you think you have cured them, but you have not.

Prevention of Subsequent Infection by Immediate Closure of Dural Opening. My views about the drainage of the subarachnoid spaces have undergone a great change. For many years I advocated and practiced opening of the dura and a continued letting out the fluid. I have reviewed 23 personal cases of cured meningitis with bacteria in the fluid on whom we had operated. Among those cases there were a large number in which ultimately an intracranial suppurative lesion recurred. They were cured of their meningitis, but subsequently succumbed to an intracranial infection.

Today, I open the cerebral spinal fluid system of the area in which the infection entered, but I immediately close this opening. I drain all the infected fluid that is adjacent to the focus, and then take a piece of mucous membrane from the lip and close the opening. Why? Because if the dural opening has any connection with the pneumatic spaces of the ear, as the dural opening does not close, there will remain a defect where infection may enter a year or more afterwards. One of our cases died of brain abscess four years after we cured the meningitis.

If the dural opening is away from any contact with the air it can be left open, but, if it is connected with the air, it must, in my opinion, be closed immediately by some tissue which is actively proliferating. Mucous membrane with submucous tissue from the lip is best. One of my associates, Dr. Richard Swain, uses a piece of facia lata; however, I do not think it is nearly as good, because it rapidly dies in the presence of suppuration and is not actively proliferating.

Toxic and Allergic Types of Nonsurgical Meningitis. There is another group of cases of bacterial meningitis which had not been described until I called attention to them at the New York Academy of Medicine, Jan. 9, 1938; they are: 1. Scarlet fever streptococcic; 2. pneumonic pneumococcic meningitis with a concomitant suppurating ear.

All these cases should be regarded and treated differently from localized bacterial meningitis of otitic or nasal origin.

The concomitant suppurative ear has to be attacked by surgery; but the meningitis should be regarded from the viewpoint of the causative scarlet fever or pneumonia.

The two last groups of nonsurgical and nondestructive types of bacterial meningitis I have named:

1. Toxemic scarlatinal meningitis, with an overflow of attenuated streptococci in the meninges.

2. Pneumococcic toxemic overflow meningitis, both of which frequently exhibit allergic phenomena and whose symptoms are due to anoxemia of the cerebral cortex.

Selected cases run a self-limited course and may end by crisis. When the overflow of bacteria is attenuated the case does not require cerebrospinal fluid drainage of any kind, but any concomitant otitis must be treated surgically.

Conclusions: I would emphasize these points: The surgical treatment of otitic meningitis requires: 1. Complete eradication of the focus of infection.

2. Operation that does less is doomed to disappointment. If the operation exposes all the bony areas of possible infection around the petrous pyramid, showing every millimeter, the focus of infection will be found and then may be removed.

3. The area of the meninges adjacent to the focus of infection which in meningitis contains infected ceerbral spinal fluid (and in many cases there are germs in this fluid, even though the lumbar puncture fluid simply gives a high cell count) should be opened, but the dura should immediately be closed again.

4. There is a group of cases that we must diagnose. These are the cases associated with scarlet fever, which I call scarlatinal toxemic meningitis, that have certain allergic symptoms.

5. There is another group of cases connected with the pneumonia and while the meningitis is limited, the same as the process in the lung, the associated otitis must be treated on the same surgical principle as if it were not a complication of either scarlet fever or of pneumonia.

6. Both of these latter types of nonsurgical overflow meningitis should be recognized as distinct entities.

SYMPOSIUM.

WHAT IS JUSTIFIABLE TO DO IN OTITIC MENINGITIS?

(d)—SULFANILAMIDE AND RELATED COMPOUNDS IN THE TREATMENT OF MENINGITIS SECONDARY TO EAR AND SINUS INFECTIONS.*†

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During the past year and a half the use of sulfanilamide has received widespread attention. It is not within the scope of this paper to discuss the history of the development and the experimental background of this chemical, nor will an attempt be made to review the extensive literature which has already been published in regard to its use. The object of this report is to present mainly the experience of the Meningitis Division of the New York Health Department with this drug in the treatment of meningitis secondary to infections of the ears and sinuses.

The scope of usefulness of sulfanilamide is not as yet completely known. It has been shown by work with animals that it is highly effective in its action on the hemolytic streptococcus, the meningococcus, the pneumococcus and the *B. Welchii*. There is also experimental work to indicate that a combination of Prontosil and specific serum has some curative action on otherwise fatal inoculations in mice with influenza bacilli. Clinical studies at present confirm the results of animal experimentation with the hemolytic streptococcus and the meningococcus. On the other hand, the results obtained clinically in pneumococcic infections have been inconclusive and in those due to the influenza bacillus very disappointing; however, our own experience indicates that sulfanilamide may be effective at times in pneumococcic meningitis. There is also clinical evidence that the chemical is of value in infections due to the gonococcus and in certain infections of the urinary tract, particularly those due to the colon

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bacillus, the staphylococcus and the Proteus group of organisms.

The mode of action of sulfanilamide is not known. Bliss and Long[1] believe that in vivo the chemical brings about a change in the micro-organisms which permits them to be phagocytized by the white blood cells. According to Osgood,[2] the major action of this drug on the hemolytic streptococcus seems to be neutralization of the toxins. At present it seems probable that under certain conditions sulfanilamide affects detrimentally the growth of bacteria and in some manner influences favorably the defense mechanisms in the body.

It has also been shown by Marshall, Emerson and Cutting[3] that this chemical has the remarkable power of penetrating all tissues. These workers have noted, following oral administration, that the drug is absorbed in about four hours, and that a maximum concentration is reached in the blood stream within four to six hours. They also demonstrated that the drug rapidly passed over into all the body fluids, including the spinal fluid, in approximately the same concentration as was found in the blood.

Sulfanilamide and its various derivatives have been used too short a time to admit of definite conclusions in regard to the relative merit of the different compounds, the best routes of administration, and the dosage. The preparations that we have used are sulfanilamide orally, Prontosil intramuscularly and at times an 0.8 per cent solution of sulfanilamide intraspinally, subcutaneously or intravenously. Recently we have used also Prontosil intraspinally and Prontosil soluble powder orally.

The compound referred to in this paper as Prontosil, the name of which is to be changed to Neo-Prontosil, is disodium 4 sulfamido -phenyl-2-azo-7-acetylamino-1-hydroxy-naphthalene-3, 6 disulfonate. This should not be confused with the earlier less soluble preparation, 4-sulfamido-2,4-diamino-azobenzene, synthesized by Mietzsch and Klarer.[4] The Prontosil has been available in solution (2.5 and 5 per cent) for parenteral use and recently also in the form of a powder for oral administration. The latter form of Prontosil is now being studied clinically by others and ourselves, but is not yet available commercially.

The relation of Prontosil to sulfanilamide is not clearly understood at the present time. As Prontosil yields only 11 gr. (0.73 gm.) of sulfanilamide per 100 cc. of the 2.5 per cent solution, it is difficult to believe that the satisfactory results observed following the daily administration of from 40 to 100 cc. of the compound is attributable solely to the sulfanilamide fraction. It would seem that Prontosil is capable of producing some other chemotherapeutic action in the body.

Recent studies by Rosenthal,[5] Barlow[6] and Raiziss, *et al.*,[7] seem to indicate that Prontosil is more effective against the hemolytic streptococcus when given orally than following subcutaneous administration. Raiziss and his associates further point out that the oral use of Prontosil yields almost as good results as the oral use of sulfanilamide. Barlow's study indicates also that Prontosil has a wider margin of safety than sulfanilamide. The oral use of prontosil seems therefore worthy of clinical trial.

In regard to dosage, it is our impression that it is not necessary to use the large doses advocated by certain workers. Indeed, recent experimental work by Osgood[2] would seem to indicate that small doses at frequent intervals are more effective than larger doses at longer intervals. As a rule, we have given 5 cc. or less of the Prontosil solution every four hours to younger children, and 10 cc. every four hours to older children and adults. In addition, from 5 to 15 gr. of sulfanilamide have been given every four to six hours. The same dosage was followed when giving Prontosil by mouth. We have felt that as a rule the combined use of Prontosil and sulfanilamide was preferable to either agent alone. At present we are inclined to use Prontosil orally, unless the patient cannot swallow or retain medication by mouth. Under these conditions it is given by injection.

We do not feel sure at present that there is any advantage in administering the sulfanilamide intraspinally. Indeed, most of our recovered cases were not treated in this manner. I have already referred to the observation of Marshall and his collaborators regarding the high concentration of sulfanilamide in the spinal fluid following oral administration; however, the 0.8 per cent sulfanilamide solution is non-irritating to the meninges and can do no harm. There may also be an advantage from a mechanical point of view in replacing a

certain amount of the fluid withdrawn. Likewise, the administration of Prontosil solution intraspinally is harmless, although it may cause a temporary rise in temperature. At present we are inclined, as a rule, to prefer Prontosil to sulfanilamide solution for intrathecal use. In this connection it should be pointed out that the pH in Prontosil is now 6.8, which is compatible with its intraspinal use.

While sulfanilamide is a therapeutic agent of great value, it has also toxic qualities of no little import. The most serious toxic manifestations are those associated with the hematopoietic system; namely, hemolytic anemia and granulocytopenia. The development of morbilliform skin rashes and fever constitute less severe reactions. Mild toxic effects are quite common and they include cyanosis, dyspnea, dizziness, nausea, headache, excitement and confusion. Certain of these reactions appear to be direct toxic effects of the drug, while others, particularly hemolytic anemia and agranulocytosis, must at present be regarded as idiosyncrasies. Fortunately, we have not yet encountered any of the more serious reactions.

With the development of the more severe toxic manifestations the drug should be withdrawn at once. We do not, however, consider cyanosis as an indication to discontinue the sulfanilamide. It should be noted that the oxygen-carrying capacity of the red blood cells is not diminished during the period of cyanosis. Dyspnea, which is due to acidosis, can usually be prevented by the routine administration of sodium bicarbonate in conjunction with the sulfanilamide. When the alkali cannot be given by mouth, one may administer a 1/6 molar sodium lactate solution by the intravenous or subcutaneous route. Since sulfanilamide is excreted more slowly than normal in conditions of impaired renal function, it should be administered with caution in such cases. It is, of course, important to make blood counts frequently, if not daily, on all patients receiving this drug.

We have used sulfanilamide in five cases that we have grouped as miscellaneous. They were all of otitic or sinus origin. In three instances the diagnosis was serous or sympathic meningitis. These patients were treated with spinal drainage and sulfanilamide. In addition, a bilateral mastoidectomy was performed on one and a petrous operation on another. These three patients recovered. It is of course difficult to evaluate the role of the drug in these recoveries. In

one instance the patient had a mastoiditis and brain abscess due to the hemolytic streptococcus. In addition to the sulfanilamide, first a mastoidectomy and then an operation on the brain abscess were done. Recovery took place. Another patient with chronic sinusitis and osteomyelitis of the skull developed meningitis. The spinal fluid showed the presence of diplococcus mucosus. The meningeal infection cleared up, following the use of antimeningococcic serum intraspinally for several days; however, the signs of meningitis reappeared two weeks later. The spinal fluid findings were confirmatory, but showed this time a different organism, staphylococcus aureus. He received a long course of treatment with sulfanilamide and bacteriophage, but died. Necropsy showed three brain abscesses, one of which was connected with the ethmoids. The meninges were practically clear.

There was a case of meningitis due to *B. Influenzae* and associated with a chronic recurring left otitis media. Sulfanilamide was used in conjunction with the specific serum. This patient died.

There were three cases of meningitis due to the nonhemolytic streptococcus, two of which were definitely of the viridans variety. In one, the meningitis was of three weeks' duration. An operation on the ethmoids and sphenoidal sinuses showed extensive involvement, with bone necrosis. Sulfanilamide was used for less than 24 hours. The patient died. The second case, in which the meningitis was secondary to a left otitis media, also ended fatally. In the third instance the meningitis was associated with a chronic sinusitis. The clinical picture was a very severe one, with marked signs of meningeal irritation, coma and left hemiplegia. Streptococcus viridans was recovered from the spinal fluid culture. Following a course of intensive sulfanilamide therapy and spinal drainage, the patient made an uneventful recovery.

I would like to refer also to two cases of meningitis in which the organisms were not definitely identified. In one instance a mastoidectomy was done for a chronic left mastoiditis and otitis. Meningitis developed seven weeks after the operation. The spinal fluid showed Gram positive diplococci on smear and staphylococci on culture. The operation was revised and pus was evacuated from the inner ear. The patient received a long course of treatment with sulfanilamide and spinal drainage, and made a complete recovery. Another

patient had meningitis following scarlet fever and mastoiditis, which was operated on. Gram positive organisms were found in the smear, but the culture was negative. This patient recovered with the use of sulfanilamide and a small amount of convalescent scarlet fever serum. This case was probably due to a hemolytic streptococcus.

We have used sulfanilamide in 32 cases of pneumococcic meningitis. Most of these cases were associated with an otitic, sinus or upper respiratory infection, or occasionally with a pneumonia. It is quite likely that in many instances an upper respiratory infection is associated with an undiagnosed sinusitis. Indeed, in one case with a clinical upper respiratory infection, an ethmoiditis was found at necropsy. An undiagnosed sinus infection may also have been present in those instances with an undetermined primary focus or in those associated with a pneumonia.

The more important data in regard to this group of cases are shown in Table I.

In this series of 32 cases, there were only four recoveries. The first was a type 31 meningitis which developed shortly after a tonsillectomy and exenteration of the right ethmoid. The second case, due to a type 29, followed within 48 hours after submucous and turbinate resections. It is probable that in these cases the primary foci were adequately drained. The third case, caused by a type 4 organism, had a double otitis media complicated by bilateral mastoiditis. A mastoidectomy was done first on the right side and a week later on the left side. Meningitis developed about a week after the second operation. In the fourth recovery the causative organism was a type 13. The primary focus could not be determined definitely in this case, although he had a slight left catarrhal otitis for one or two days. This patient ran a long course, approximately five weeks, before the spinal fluid became sterile. There was also a short relapse late in his convalescence. It should be noted that these four patients who recovered had persistently negative blood cultures.

As shown in Table I, none of the recovered patients received serum.

Among the fatal cases. there was a great variety of types of pneumococci with a slight preponderance of types 1 and 3.

TABLE I.

Cases of Meningitis Due to the Pneumococcus.

Case	Sex	Age, Years	Primary Source of Infection	Type	Blood Culture	Treatment	Result	Remarks
1	F	52	Right otitis.	3	—	Spinal drainage, sulfanilamide, mastoidectomy.	Died	
2	M	50	Tonsils, ethmoids.	31	Negative	Tonsillectomy, ethmoidectomy, spinal drainage, sulfanilamide.	Recovered	Onset of meningitis within 48 hours after operation.
3	F	65	Pneumonia?	13	—	Spinal drainage, sulfanilamide.	Died	Had marked arteriosclerosis and hypertension.
4	M	5 mos.	Bilateral otitis.	3	—	Spinal drainage, sulfanilamide.	Died	
5	M	3 mos.	Upper respiratory infection.	5	—	Spinal drainage, sulfanilamide.	Died	
6	F	47	Upper respiratory infection.	Beyond 32	—	Spinal drainage, sulfanilamide.	Died	Day of onset received spinal anesthesia.
7	M	8 mos.	Undetermined.	7	—	Spinal drainage, sulfanilamide, antipneumococcic serum intraspinally and intravenously.	Died	
8	M	22	Nasal cavity.	29	Negative	Submucous and turbinate resections, spinal drainage, sulfanilamide.	Recovered	Onset of meningitis within 48 hours after operation.
9	F	10	Upper respiratory infection.	6	Positive	Spinal drainage, sulfanilamide, antimeningococcic and antipneumococcic serums.	Died	
10	M	9	Bilateral mastoiditis.	1	Negative	Spinal drainage, sulfanilamide, antimeningococcic and antipneumococcic serums, right mastoidectomy.	Died	Developed dyspnea and pulmonary edema. Probably had pneumonia. Also signs of left mastoiditis.

TABLE I.

Cases of Meningitis Due to the Pneumococcus (Continued).

Case	Sex	Age, Years	Primary Source of Infection	Type	Blood Culture	Treatment	Result	Remarks
11	M	8	Left otitis and mastoiditis.	1	Negative	Spinal drainage, sulfanilamide, antipneumococcic serum intraspinally and intravenously, bilateral mastoidectomy, transfusion.	Died	
12	F	59	Upper respiratory infection.	6	—	Spinal drainage, sulfanilamide.	Died	Onset with convulsions and coma. Fulminating case.
13	M	1	Bilateral otitis.	31	—	Spinal and perivascular drainage, antistreptococcic serum.	Died	X-ray mastoids negative.
14	F	5	Bilateral otitis.	4	Negative	Spinal drainage, sulfanilamide, bilateral mastoidectomy.	Recovered	Meningitis developed after second operation. No clinical, but X-ray evidence of mastoiditis. Both mastoids necrotic at operation.
15	F	7	Nasopharyngitis.	6	Positive	Spinal drainage, sulfanilamide.	Died	
16	F	56	Bilateral otitis and mastoiditis.	3	—	Spinal drainage, sulfanilamide, bilateral mastoidectomy one month previously.	Died	Fulminating case. Was diabetic. Sulfanilamide used for about 10 hours before death.
17	M	15	Bilateral otitis.	1	—	Spinal drainage, sulfanilamide, antipneumococcic serum intravenously and intraspinally, bilateral mastoidectomy.	Died	Showed temporary improvement. Necropsy showed extensive basilar meningitis.
18	F	28	Right otitis.	23	—	Spinal drainage, sulfanilamide, antimeningococcic serum.	Died	

TABLE I.

Cases of Meningitis Due to the Pneumococcus (Continued).

Case	Sex	Age, Years	Primary Source of Infection	Type	Blood Culture	Treatment	Result	Remarks
19	M	11	Chronic sinusitis.	3	Negative	Spinal drainage, sulfanilamide, antimeningococcic and antipneumococcic rabbit serums.	Died	Right mastoidectomy 6 years ago. Showed temporary improvement.
20	M	7 mos.	Upper respiratory infection. Ethmoiditis. (Postmortem.)	6	Positive	Spinal drainage, sulfanilamide, antimeningococcic and antipneumococcic serums. One cisternal tap.	Died	Patient died shortly after cisternal tap. Necropsy showed purulent meningitis, subarachnoid hemorrhage over medulla and base, and purulent ethmoiditis. The ethmoiditis was not found clinically.
21	F	4½	Upper respiratory infection.	18	—	Spinal drainage, sulfanilamide.	Died	X-ray of lungs and mastoids negative.
22	F	5 mos.	Pneumonia and left catarrhal otitis.	6	—	Spinal drainage, sulfanilamide, antipneumococcic serum.	Died	Meningitis was terminal.
23	M	31	Undetermined.	10	Positive	Spinal drainage, sulfanilamide, Prontosil intraspinally.	Died	Fulminating case, with coma shortly after onset.
24	M	57	Upper respiratory infection. Pneumonia.	1	Positive	Spinal drainage, antimeningococcic and antipneumococcic serums intraspinally and intravenously, 1 dose of Prontosil intramuscularly.	Died	Meningitis terminal.
25	M	15	Catarrhal otitis?	13	Negative	Spinal drainage, sulfanilamide, transfusions.	Recovered	One month after onset showed marked improvement, although spinal fluid cultures remained positive for another week. Was apparently well for one month, when he had a short relapse. Finally recovered.
26	M	35	Sinusitis.	18	—	Spinal drainage Prontosil intramuscularly.	Died	Prontosil used only for about 24 hours.

TABLE I.

Cases of Meningitis Due to the Pneumococcus (Continued).

Case	Sex	Age, Years	Primary Source of Infection	Type	Blood Culture	Treatment	Result	Remarks
27	M	3½	Ethmoiditis.	1	—	Spinal drainage, sulfanilamide, antipneumococcic serum and Prontosil intraspinally.	Died	Had also varicella.
28	M	10	Sinusitis and mastoiditis.	3	Negative	Spinal drainage, sulfanilamide, Prontosil intramuscularly, left mastoidectomy.	Died	X-ray positive for left mastoid, but negative for right mastoid. After mastoidectomy he improved and the spinal fluid sterile for three weeks. Then active meningitis again. Necropsy showed infection in sphenoid, ethmoid and mastoid, all on right side.
29	M	53	Upper respiratory infection. Sinusitis?	8	Positive	Spinal drainage, sulfanilamide, antipneumococcic rabbit serum intraspinally and intravenously, Prontosil intraspinally.	Died	Was in coma.
30	F	16	Sinusitis.	6	Negative	Spinal drainage, sulfanilamide, Prontosil intraspinally and orally, rabbit antipneumococcic serum intraspinally.	Under observation	Showing improvement.
31	M	48	Sinusitis.	3	Negative	Spinal drainage, sulfanilamide, submucous resection, ethmoidectomy, sphenoidectomy, antrotomy.	Died	Meningitis developed within 24 hours after the operations. Showed marked temporary improvement. Developed hemiplegia before death.
32	F	20	Sinusitis.	7	—	Spinal drainage, sulfanilamide, Prontosil intramuscularly.	Under observation	

In most of these cases it was impossible to diagnose during life the focal infection with sufficient definiteness to warrant an operation. It is very likely that the failure to eradicate the primary foci may have been an important factor in the unfavorable results.

While the clinical results with sulfanilamide in pneumococcic meningitis are far from satisfactory, they are nevertheless encouraging, and indeed, are favorable when compared to our former case fatality, which had been 100 per cent. Furthermore, in several of the fatal cases the clinical course was markedly prolonged, at times five or six weeks, with long intervals of improvement. It is possible, perhaps even probable, that proper removal of the primary foci might have led to recovery in these instances.

The use of sulfanilamide in cases of hemolytic streptococcus meningitis has yielded quite astounding results. We have had 26 cases, with 21 recoveries and five deaths.

The diagnosis in these cases was definitely established by recovering the hemolytic streptococcus from the spinal fluid culture. In this connection it is important to stress that certain strains of this organism hemolyze only horse blood, and in media which do not contain sugar. Unless this is borne in mind, the hemolytic streptococcus will occasionally be misinterpreted as the nonhemolytic variety.

Table II shows the more important points in regard to this group of cases.

It will be noted that all but two of these cases have been secondary to infections of the ears or mastoids. One case was associated with a pansinusitis and another followed a throat infection.

In reviewing the records of the five fatal cases it may be noted that sulfanilamide was used less than 12 hours in two instances. In another case, the chemical was used less than 24 hours before death. This patient showed at necropsy a very extensive pansinusitis and a sarcoma of the pituitary body. In the fourth case the necropsy showed extensive thrombosis of the left lateral and transverse sinuses and herniation of the cerebellum through the operative wound. The fifth fatal case represents a definite failure of sulfanilamide therapy for which I have no adequate explanation.

A comparison of these results with those previously obtained by us is indeed striking. During the period of nearly 28 years that the meningitis division has been in existence, the case fatality in hemolytic streptococcic meningitis has been uniformly very high. Up to the end of 1936, we had seen 274 cases of all kinds of streptococcic meningitis, most of which have been of the hemolytic variety. Of these cases, only 15 recovered, of which nine were definitely caused by the hemolytic streptococcus. There was a tenth recovery in which there was a mixed infection of the meningococcus and the hemolytic streptococcus. Three of these recoveries were in patients in whom the meningitis developed following scarlet fever. In two instances the antiscarlatinal serum was used, and in the third large amounts of convalescent serum and small amounts of Prontosil and Prontylin.

The question is sometimes raised as to the possibility that the hemolytic streptococcus may recently have become less virulent. Such a radical and sudden change seems to us most improbable. During the year 1936, we saw 20 cases of hemolytic streptococcic meningitis, all of which died with the exception of one following scarlet fever, to which I have already referred.

It is important in all forms of meningitis to adequately drain the subarachnoid space. In our experience, the more radical methods of establishing drainage, such as laminectomy, or trephining the cisterna magna, have no advantage over repeated lumbar punctures or cisternal or ventricular puncture if block occurs. Indeed, establishing permanent drainage may be a disadvantage if serum or chemicals are to be used intraspinally. We do not recommend the use of forced spinal or perivascular drainage.

The prompt and complete eradication of the primary foci of infection is of particular importance in the forms of meningitis under consideration. It cannot be too strongly emphasized that, not infrequently, a severe mastoiditis may fail to present clinical symptoms and occasionally even Roentgenographic evidence of involvement. Several such instances are shown in Tables I and II. Of course, a patient will occasionally recover even if the focal infection is not removed. It may be noted, in Table II, that eight patients recovered without an operation. But, as a rule, it is too great a risk not to eradicate the primary focus.

TABLE II.

Cases of Meningitis Due to the Hemolytic Streptococcus.

Case	Sex	Age, Years	Primary Source of Infection	Treatment	Result	Remarks
1	F	21	Right maxillary sinusitis. Right otitis with mastoiditis.	Drainage antrum; mastoidectomy, spinal drainage, sulfanilamide, convalescent scarlet fever serum.	Recovered	Only a small amount of serum was given.
2	F	7	Left otitis with mastoiditis.	Mastoidectomy, spinal drainage, sulfanilamide.	Recovered	
3	M	5½	Left otitis.	Antimeningococcic serum (four doses), spinal drainage, sulfanilamide.	Recovered	
4	F	43	Right otitis.	Spinal drainage, sulfanilamide.	Recovered	
5	F	3½	Left otitis.	Spinal drainage, sulfanilamide.	Recovered	
6	F	11	Right otitis with mastoiditis.	Spinal drainage, sulfanilamide.	Recovered	Refused operation and mastoiditis persisted after recovery from meningitis.
7	F	2	Double otitis with right mastoiditis, following scarlet fever.	Right mastoidectomy with ligation right jugular, spinal drainage, sulfanilamide, convalescent scarlet fever serum.	Died	Meningitis appeared seven days after operation. Necropsy showed herniation of cerebellum and thrombosis of left lateral and transverse sinuses, and of left jugular.
8	M	5½	Left otitis with mastoiditis, following pneumonia.	Mastoidectomy, antimeningococcic serum, spinal drainage, sulfanilamide.	Recovered	
9	M	8	Right otitis with mastoiditis.	Mastoidectomy one month prior to meningitis, spinal drainage, sulfanilamide.	Recovered	Mastoidectomy done about one month before onset of meningitis. Spinal fluid showed positive culture for 14 successive days.

TABLE II.

Cases of Meningitis Due to the Hemolytic Streptococcus (Continued).

Case	Sex	Age, Years	Primary Source of Infection	Treatment	Result	Remarks
10	F	4	Left otitis with mastoiditis and later right otitis.	Left mastoidectomy, spinal drainage, sulfanilamide.	Recovered	At operation, left mastoid found necrotic, although no clinical signs.
11	M	35	Pansinusitis.	Antimeningococcic serum, spinal drainage, sulfanilamide.	Died	Sulfanilamide was used for less than 24 hours. Necropsy showed pansinusitis and sarcoma of pituitary.
12	F	12	Left otitis with mastoiditis.	Mastoidectomy, spinal drainage, sulfanilamide, one dose antimeningococcic serum.	Recovered	At operation, mastoid found necrotic, although no clinical signs.
13	F	10 mos.	Double otitis with double mastoiditis.	Mastoidectomy, spinal drainage, sulfanilamide.	Died	There was also clinical evidence of brain abscess. At operation, dural plate found nearly destroyed. Sulfanilamide used for less than 12 hours.
14	F	8	Left otitis with mastoiditis.	Mastoidectomy, spinal drainage, convalescent scarlet fever serum, sulfanilamide.	Recovered	At operation, mastoid found necrotic, although no clinical signs.
15	F	6½	Right otitis with mastoiditis.	Mastoidectomy, spinal drainage, sulfanilamide.	Died	Necropsy showed meningitis, but no localized suppuration.
16	M	4	Following measles, first left otitis with mastoiditis and later right otitis with mastoiditis.	First left mastoidectomy and later right mastoidectomy, spinal drainage, sulfanilamide.	Recovered	Meningitis appeared 12 days after left mastoidectomy. At this time a right mastoidectomy done.

TABLE II.

Cases of Meningitis Due to the Hemolytic Streptococcus (Continued).

Case	Sex	Age, Years	Primary Source of Infection	Treatment	Result	Remarks
17	M	7	Double otitis with double mastoiditis following scarlet fever.	First left mastoidectomy and three weeks later, right mastoidectomy, spinal drainage, sulfanilamide.	Recovered	Onset of meningitis was about 12 days after the second mastoidectomy.
18	F	11 mos.	Upper respiratory infection.	One spinal drainage, antimeningococcic serum, sulfanilamide.	Died	Death in less than 12 hours after receiving sulfanilamide.
19	F	6	Double otitis, double mastoiditis.	Double mastoidectomy, spinal drainage, sulfanilamide.	Recovered	
20	F	8	Chronic left otitis.	Spinal drainage, sulfanilamide orally, Prontosil intramuscularly and intraspinally, mastoidectomy.	Recovered	Mastoid at operation showed granulation, but no pus. Blood culture sterile.
21	M	2	Right otitis.	Spinal drainage, sulfanilamide.	Recovered	No signs of mastoiditis.
22	M	15	Bilateral chronic otitis.	Spinal drainage, sulfanilamide, left mastoidectomy and two months later right mastoidectomy.	Recovered	Marked improvement after first operation, and complete recovery followed second operation.
23	M	7	Right otitis and mastoiditis.	Spinal drainage, sulfanilamide, mastoidectomy.	Recovered	
24	F	12	Right otitis.	Spinal drainage, sulfanilamide orally and intravenously.	Recovered	
25	F	11	Right otitis.	Spinal drainage, Prontosil intramuscularly and intraspinally. Prontosil orally.	Recovered	
26	F	7	Throat infection.	Spinal drainage, Prontosil intramuscularly, sulfanilamide orally.	Recovered	X-ray of mastoids and sinuses negative.

There can be little question that the development of sulfanilamide is one of the major achievements of modern therapeutics. It has stimulated a genuine interest in the subject of chemotherapy. Research work is being carried on intensively in an effort to prepare better compounds. Indeed, the future looks bright for even greater progress in this revolutionary field of chemotherapy of bacterial infections.

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DISCUSSION.

Dr. Josephine B. Neal (New York): It is a little difficult to discuss a paper given by Dr. Appelbaum as he covers all the points so thoroughly and clearly. Anything that I may say will be more or less in the way of emphasizing points that he has already brought out.

In the first place, mention has been made of the penetration of all of the tissues, even of bone, by sulfanilamide. I have often questioned the efficacy of the action of sulfanilamide on infections in bony tissues, as in mastoids or sinuses. The reason for this is that I have seen, in several instances, the persistence of the clinical course in spite of the use of sulfanilamide in adequate doses.

This leads naturally to emphasizing the necessity of an early diagnosis and complete removal of all foci of infection. For example, I recall a case that improved greatly after one mastoid was removed. The improvement, however, did not go on to recovery until after the second mastoid was operated. Of course, a mastoiditis or sinusitis may subside spontaneously. There is always the possibility in these cases that these foci may later light up, with a resulting meningitis or brain abscess.

When block occurs in children with an open fontanelle, we greatly prefer a ventricular puncture to a cisternal puncture. There are two reasons for this: one is that the ventricular puncture is more likely to be effective since the adhesions may be below the cistern; the other is that ventricular puncture is far safer than cisternal punctures. Fortunately, in the experience of our division, no fatalities have followed a cisternal puncture, but in the course of conversation, probably 25 or 30 such fatalities have been described to us, occurring in meningitis, although the operator was experienced. A cisternal puncture in meningitis offers quite different problems from a cisternal puncture in syphilis of the central nervous system, where there are no adhesions and where the blood vessels are not congested.

Some years ago, we tried perivascular drainage or, as it was then called, forced spinal drainage. We have never seen an instance in meningitis in which this procedure seemed to be of value, and we have seen several instances in which it was definitely proved at necropsy that it had unfavorable effects.

The results of sulfanilamide in the treatment of hemolytic streptococcic meningitis have seemed to us to be really marvelous. It is true that, in our experience, there have been a few deaths, but most of these were on the basis of attendant circumstances rather than on the lack of efficacy of sulfanilamide, and, for that matter, what medication gives 100 per cent recoveries?

I can only repeat Dr. Appelbaum's praise of the value of sulfanilamide, and point out again the necessity that reliable products be used.

In closing, I would like to challenge the members of this Society to find methods by which the diagnosis of mastoiditis and sinusitis can be made earlier.

SURGICAL TECHNIQUE FOR THE CONSERVATION OF THE HEARING IN CHRONIC MASTOIDITIS.*

DR. J. MORRISSET SMITH, New York.

The conservation of the hearing in chronic mastoiditis presents a very complex problem. It is difficult, first, because in all of these cases there is a permanent destruction of part of the conducting apparatus; next, due to the sclerosis in the average bone, there is no way of determining before operation the true extent of the destruction; and, finally, there is the ever-present necessity for removing the danger to the life of the patient, regardless of the effect on the hearing.

The average chronic mastoid infection requiring operation usually has a continuous foul-smelling discharge persisting in spite of local treatment and medication, indicating definite evidence of bone destruction.

If the structural anatomy involved is examined the infection will be found locked in a bony vault by the ossicles, the vault consisting of the attic, antrum and mastoid cavity, the lock, the malleus and incus or their remnants, plus the infected granulation tissue surrounding them.

The marked variation in the normal anatomy of the temporal bone, from the almost solid infantile type without any mastoid cells, a small antrum, attic and middle ear, to the extremely pneumatic with cells extending throughout the temporal bone, including the petrous pyramid, makes it readily apparent that there must be a very wide variation in the degree of necrosis encountered in the different individuals.

For this reason, the writer believes it is impossible to deal with all of these cases by the use of one type of operation, as many of the otologists have advocated in the past, and that the only successful way to manage all of them is by the use of a graduated surgical technique applicable to the degree of necrosis encountered in the different cases.

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We have all seen patients without a history of a previous operation where the drum and malleus have been destroyed by the disease, but with a clean, dry middle ear and good, practical hearing; by this I mean sufficient hearing to enable the individual to earn a living following an average vocation.

The lesson to be learned from this is that, even though it may be necessary to remove the drum and ossicles, it is possible to conserve or even improve the hearing if we can secure and maintain a clean internal wall of the middle ear, free from granulation tissue.

Dealing with the problem as a whole, my first effort consisted in the performance of a large number of radical mastoid operations. I found that, regardless of the thoroughness of the operative technique and the after-treatment, in many instances a layer of granulation tissue formed over the internal wall of the middle ear, even though it was later apparently absorbed and the ear became dry, the resulting layer of fibrous tissue so seriously impaired the hearing that it was of little or no practical value to the patient.

I next attempted to avoid this in cases requiring the removal of the malleus, incus and drum by devising an operation, which I called "The New Radical Operation" and described its technique in the *Medical Journal and Record*, of April 3, 1929. This I still employ, with a slight modification of the surgical management of the middle ear.

The ultimate objective in the management of these cases is, therefore, first to remove the danger and then allow as much of the conducting apparatus to remain as possible in those cases in which it is consistent with the safety of the individual; next, where it is necessary to remove the ossicles, to attempt to secure and maintain a clean internal wall of the middle ear so that the sounds may have an untrammeled pathway to the outer wall of the internal ear, especially in the presence of a bilateral infection, where the conservation of hearing may be of vital importance to the patient.

Unless an emergency exists, the tonsils and adenoids should be thoroughly removed and any existing nasal infection or obstruction should be corrected before operation.

The graduated surgical technique employed in the management of these cases is as follows: "The Complete Simple Mas-

toid Operation"; "The Complete Simple Mastoid Operation Plus Removal of the Incus"; "The New Radical Operation"; "The Complete Radical Operation."

THE COMPLETE SIMPLE MASTOID OPERATION.

This is the most conservative means of preserving the hearing in chronic mastoiditis. It is indicated in the cases where the removal of the drum and ossicles is not necessary.

The Technique: The usual incision and exposure of the cortex is made, the cortex is removed and the contents of the mastoid cavity exenterated to healthy plate in both the middle and posterior fossae. The mastoid antrum is then widely opened and the dissection of the bone at the root of the zygoma is carried forward until the short process of the incus is thoroughly exposed at its attachment just external to the horizontal semicircular canal, all the accessible granulation tissue in the posterior part of the additus is removed. Peroxide and salt solution are repeatedly used to flush the cavity during the operation.

A cigarette drain is now inserted directly into the antrum from behind and this space allowed to remain open, with a partial closure of the wound below from the tip. This allows direct access to the attic region for daily inspection and irrigations during the after-treatment. The mastoid wound is allowed to fill in and heal with healthy granulation tissues in the simple mastoid. The external canal is packed and cleansed daily with peroxide and alcohol for seven days, then the packing is discontinued and peroxide and boric acid irrigations are used three times daily, followed by 95 per cent alcohol drops.

All the accessible granulation tissue of polypi in the middle ear should be removed through the external canal either before or at the time of the operation.

Properly performed, this operation eradicates the diseased bone in the mastoid, the antrum and the posterior part of the additus, leaving only a small amount of granulation tissue in the attic or upper part of the middle ear. The normal external auditory canal is preserved and cutting a flap is avoided. It will frequently result in a dry ear, with the preservation of valuable hearing, and is especially indicated in young children.

THE COMPLETE SIMPLE MASTOID PLUS THE REMOVAL OF THE INCUS.

There will be some cases in which the removal of the incus will greatly facilitate the removal of the granulations from the attic and improve the chances of recovery.

Technique: The steps of the complete simple mastoid operation are performed in detail as described above, then the point of a Eustachian curette is inserted under the incus and it is dislocated backward into the antrum and removed. The head of the malleus is now visible and allowed to remain undisturbed, all the accessible granulations are carefully removed from the attic, remembering not to remove the stapes or injure the facial nerve by making pressure over the tympanic portion of the Fallopian canal. The postoperative care described above is carried out in every detail. This leaves the remainder of the drum and the malleus in their normal position. The mastoid wound heals as in the simple operation. It is remarkable how little effect the removal of the incus has upon the hearing.

THE NEW RADICAL OPERATION.

The third step in the graduated technique I termed "The New Radical Operation" for want of a better name. It is devised to care for some of the cases requiring removal of the malleus and incus and still avoid a complete radical operation.

It is especially indicated in cases where the hearing in the other ear may have been lost or in the presence of an extensive bilateral infection, where the preservation of the hearing may again be vitally important. I have modified the technique of the exenteration of the contents of the middle ear since the original operation was described. It is now as follows:

The same incision, exposure of the cortex, exenteration of the mastoid and removal of the incus as described above is performed. A small part of the bridge next to the incus is removed from within outward to enlarge the additus. Then the Eustachian currette is again used to dislocate the malleus and it is removed with a small forceps. This completes the opening of the tubular space connecting the mastoid cavity and the external auditory canal and permits a through-and-through irrigation from the mastoid antrum through the attic

and middle ear and out through the external auditory canal. Salt solution and peroxide are repeatedly used to remove the debris and granulations, followed by 1/1000 adrenalin.

The membranous canal is not disturbed; a speculum is now inserted in the external canal and a ring curette placed in the center of the promontory and the granulations may be curetted downward, backward and forward, taking care not to make pressure upward over the facial nerve and the stapes. Remnants of the drum may be removed; the annulus is allowed to remain. Adrenalin, peroxide and saline irrigations are continuously used to facilitate the cleansing of the tympanum. A silver probe may now be gently used to properly locate the Eustachian tube, followed by a Eustachian curette. Alcohol is now freely used in the tympanic cavity and attic.

The operative field now consists of a clean mastoid, antrum and attic, with the malleus, incus, most of the drum and granulation tissue removed from the middle ear. The posterior bony and membranous canal remain in their normal position. A small piece of folded rubber tissue is now inserted into the opening between the middle ear and mastoid antrum; the canal and middle ear packed with a narrow cigarette drain. A cigarette drain is also inserted directly into the antrum behind, as described in previous operations. A daily through-and-through irrigation of the cavity with peroxide and salt solution, with the use of alcohol in the middle ear, is absolutely essential to aid Nature in overcoming the remaining infection in the healing process. A dry, clean middle ear with a healed mastoid wound behind is the objective.

In the original description of this operation, the removal of the contents of the tympanum was accomplished by separating the membranous canal and replacing it at the conclusion of the operation. The denuded bone at the inner part of the bony canal resulting from this and the removal of the annulus tympanus made it too difficult to control the formation of granulation tissue in the tympanum during the after-treatment; for this reason, the membranous canal and the annulus are allowed to remain undisturbed, as described above.

In some instances, where the middle ear is packed with dense granulations, better results may be obtained by following the original technique.

This operation will be successful in many cases upon which complete radical operations have been performed in the past.

THE COMPLETE RADICAL OPERATION.

The fourth and final step: The extensive necrosis encountered in a small number of the chronic infections will permit nothing short of a complete radical operation. It is chiefly indicated where one of the above procedures has failed to check the progress of the disease or where there is definite or suspected evidence of an internal ear involvement, intracranial extension of the disease, facial paralysis, petrositis, certain cases of extensive cholesteatoma, etc. It makes possible the most complete eradication of the disease in the tympanum, attic and mastoid by removing the posterior bony canal and converting these spaces into one large cavity.

It is very definitely indicated in a small percentage of the chronic infections and cannot be replaced by any of the modifications.

A detailed description of this operation by the writer may be found in the *Transactions of the American Otological Society*, May 19, 1930.

The ordinary modified radical may be included in the above technique; I prefer to use the complete simple operation in those cases where the ossicles and the remnants of the drum may be allowed to remain. It is a useful procedure, its technique well known, and may be employed by those who prefer it to the above operation. I like to use it in the cases with a small attic perforation in which most of the drum and ossicles are still in good condition but there is a cholesteatoma present. Due to the tendency of the cholesteatoma to recur, lowering the facial ridge to the annulus and cutting a flap gives permanent access to the antrum and mastoid spaces.

The ossicilectomy is not used, since it leaves both the operator and the patient totally in the dark concerning the extent of the necrosis beyond the attic. The patient deserves the safety insurance that goes with the inspection and eradication of the disease in the antrum and mastoid cavities.

There is no royal road to success in dealing with this problem. There is already present a permanent loss of part of the hearing, due to a destruction of part of the conducting

apparatus. If this loss should be of the nature of that due to catarrhal deafness or due to the involvement of the perception apparatus, no improvement may be expected. In other instances, it will be hard to decide whether to allow the conducting apparatus to remain or whether it is best to remove it; at other times a great deal of difficulty will be encountered in keeping the middle ear free from granulation tissue; some of the attempts will fail, necessitating a complete radical operation.

SUMMARY.

The first objective is the removal of the danger to the life of the patient.

The next, the conservation of as much of the conducting apparatus as is consistent with the safety of the individual. This is accomplished by steps No. 1 and 2.

The third is the attempt to secure a clean, dry internal wall of the middle ear, free from granulation tissue, where the removal of the malleus, incus and drum are necessary. Step No. 3 is indicated here.

The fourth objective is the complete exenteration of the contents of the middle ear, attic, antrum and mastoid, indicated by the extensive necrosis, and is accomplished by the complete radical operation.

The technique as a whole, including the modified radical, offers a selective operation capable of adequately caring for any degree of necrosis encountered in chronic mastoiditis, exclusive of a petrositis or an intracranial extension of the infection.

The type of operation indicated must necessarily be dictated by the degree of necrosis encountered in the individual case. If the above technique is carefully followed, it will not only conserve but improve the hearing in many instances.

CASE REPORTS.

COMPLETE SIMPLE PLUS REMOVAL OF INCUS.

Case 1: Female, age 20 years. Chronic mastoiditis right, several years' duration; part of drum destroyed; foul discharge; not much granulation middle ear. Operation, Aug. 25, 1937. Sclerotic cortex no mastoid cavity, antrum attic filled with pus and granulation tissue, incus destroyed, malleus and part of drum allowed to remain. Through-and-through irrigation. Mastoid wound healed, ear dry. Hearing before operation, right ear 40 per cent loss; five months after operation, 20 per cent loss. Twenty per cent improvement.

NEW RADICAL OPERATION.

Case 2: Male, age 20 years. Chronic mastoiditis 15 years. Examination middle ear completely filled with granulations and pus; no drum visible. Operation, Feb. 17, 1938. Sclerotic bone with fistula found leading into mastoid antrum; no mastoid cells; antrum attic and middle ear filled with granulations and pus; no ossicles found; some cholesteatoma. Entire contents of middle ear attic and antrum exenterated; membranous canal not disturbed; cavity irrigated through-and-through repeatedly with peroxide and saline solution.; cigarette drain behind and in middle ear cavity syringed through-and-through for about eight weeks; posterior wound then healed; middle ear about dry April 26, 1938. Hearing before operation, 52 per cent loss; two and one-half months after operation, 38 per cent loss.

Comment: Extensive destruction prevented much improvement; surprised at 14 per cent gain.

COMPLETE RADICAL OPERATION.

Case 3: Male, age 40 years. Chronic mastoiditis left since infancy. Lately slightly dizzy, headache, occasional nausea. Examination complete sagging, external canal narrowed down to small slit, scant very foul discharge, complete loss of hearing, tuning fork referred to opposite ear. Immediate operation sclerotic cortex with large perforation in external auditory canal, entire mastoid cavity and middle ear exenterated by large cholesteatoma; facial ridge destroyed; facial nerve exposed just below horizontal canal; fistula found in horizontal canal. Complete radical operation performed; labyrinth not disturbed; ear completely healed and radical cavity dermatized in 12 weeks.

Comment: Extensive destruction necessitated a complete radical operation. Total loss of hearing; operation had no effect upon it.

COMPLETE SIMPLE PLUS REMOVAL OF INCUS.

Case 4: Male, age 25 years. Double chronic mastoiditis since infancy; small attic perforation both ears, with scant foul smelling discharge; cholesteatoma demonstrable both side. Operation, left ear April 1, 1937. Sclerotic bone, no cells; antrum contained granulations and cholesteatoma; incus removed; cholesteatoma removed from attic; through-and-through irrigation. Malleus and drum allowed to remain. Ear healed, middle ear dry, right ear unoperated. Audiogram left, preoperative, 25 per cent loss. Audiogram left, postoperative, 33 per cent loss eight months later.

Comment: Small attic perforation, with good drum and hearing, and cholesteatoma present a complex problem due to tendency of cholesteatoma to recur. I believe regular modified radical best procedure and will advise it for right ear.

NEW RADICAL OPERATION.

Case 5: Male, age 28 years. Double chronic mastoiditis since childhood; foul smelling discharge; granulations in both middle ears and destruction of most of drum on both sides. New radical operation, with removal of remnants of drum and ossicles right ear Aug. 5, 1937; left ear Sept. 29, 1937. Bridge allowed to remain, no flap cut. Both healed behind as in simple. Hearing before operation, right, 52 per cent loss; left, 50 per cent loss. Patient's hearing prevented his obtaining position. Hearing Dec. 12, 1937, right, 21 per cent loss; left, 38 per cent loss. Improved 31 per cent right ear, dry; 12 per cent left, slight mucoid discharge. Secured position and disappeared, preventing further follow-

up. This method of dealing with an extensive double chronic suppurative mastoiditis is decidedly better than a double radical operation.

NEW RADICAL MASTOID OPERATION.

Case 6: Female, age 19 years. Chronic mastoiditis left since childhood. New radical operation, April 9, 1936. Sclerotic cortex antrum attic filled with cholesteatoma; incus destroyed; remnants of drum and malleus removed; through-and-through irrigation; mastoid wound healed. Ear did not become dry until four months later. Examined Dec. 16, 1937. Recurrence of cholesteatoma, with exenteration of bridge and bone in attic; now has regular radical cavity. Record of hearing before operation not found. Audiometer Dec. 16, 1937, showed only 22 per cent loss in operated ear.

Comment: Cholesteatoma very apt to recur. Would perform radical operation in future if there is normal hearing in the other ear.

NEW RADICAL OPERATION.

Case 7: Male, age 34 years. O. M. P. C. residual right ear. Chronic mastoiditis left ear several years; very foul smelling discharge; drum destroyed, with some granulation middle ear. New radical operation five years ago; sclerotic bone thick; cortex, antrum and attic filled with cholesteatoma; incus destroyed; piece of malleus and annulus removed; bridge allowed to remain. Posterior wound healed, middle ear clean, slight mucoid discharge. Patient disappeared. Examination five years later, March 15, 1937, foul discharge with polyp in attic. Polyp removed, cholesteatoma found back of it. Cavity treated four weeks now; slight mucoid discharge, no odor. Cholesteatoma disappeared; hearing five years after operation, audiometer, 30 per cent loss right, 34 per cent left. Middle ear clean, free from granulations.

Comment: Audiometer before operation, right ear, 36 per cent loss; five years later, 30 per cent loss. Left ear, 37 per cent loss; five years later, 34 per cent loss. Patient's hearing slightly better five years after operation. Cholesteatoma may recur.

COMPLETE SIMPLE PLUS REMOVAL OF INCUS.

Case 8: Female, age 7 years. Chronic mastoiditis right, four years' duration; very foul discharge. Part of drum destroyed and some granulation tissue middle ear. Operation: cortex removed; large perisinus abscess found region of knee of sinus; extensive excavation in antrum and attic by disease; incus destroyed; large amount of cholesteatoma removed from attic. Solution syringed through-and-through both ways. Remnants of drum and malleus allowed to remain. Operation, complete simple, plus removal of incus. Audiogram before operation, 32 per cent loss. Ten months after operation, 24 per cent loss. Wound healed, ear dry. No preoperative symptoms or evidence of the large perisinus abscess disclosed by the operation. Danger removed; slight improvement in hearing.

These cases give some idea of the difficulties encountered in dealing with this disease, and while the results are far from perfect, I believe they definitely point the way to a progressive improvement in their management.

123 East 53rd Street.

DISCUSSION.

DR. HAROLD I. LILLIE (Rochester, Minn.): The adaptation of surgical measures to the needs of the pathologic process encountered long has been considered good surgical judgment. The adaptation of all pathological processes to certain technical procedures is not considered to be good surgical judgment. Dr. Smith, very apparently, is an exponent of the first proposition.

All of us, no doubt, have observed cases of chronic suppurative otitis media in which a cure has been effected by natural reparative processes. All the surgeon can do is to attempt to help the natural reparative processes accomplish their aim by removing the obstacle that prevents the successful accomplishment of the natural tendency. The recognition of that obstacle is surgical judgment.

The surgeon, in choosing certain technical proceedings for combating a given pathologic process, must know in advance of the operation the probable character and extent of the pathologic process. Such information comes only from taking into consideration the history of the lesion and observing its effect on the involved tissues. The history often may give more important information than an examination.

Chronic suppurative otitis media may be classified empirically into clinical groups that are accurate enough so that all otologists would recognize immediately what group was being considered in a discussion. When it is strongly suspected that cholesteatoma is present, it is my opinion that some form of radical operation must be used to order to prevent recurrence of the cholesteatoma; otherwise, the purpose of the operation is defeated.

I find myself in close agreement with Dr. Smith about operative measures through the external auditory canal; however, I have observed pathologic processes in which the posterior and superior bony walls of the external auditory canal had been destroyed by the disease and the cavity within the mastoid was separated from the meatus only by the membranous canal. The intra-aural removal of the overlying membranous meatus and postoperative treatment were sufficient to effect a cure. If the bony wall has not been destroyed, I know of no intra-aural surgical measure that would be adequate.

In cases of bilateral involvement of the ears, conseravtion of the hearing becomes an added responsibility; however, if the pathologic lesion is such that life is threatened if the progress of the lesion is not checked, the conservation of hearing is of secondary consideration. In many cases, the hearing may be greatly improved by the use of an artificial prothesis; curiously enough, the prosthesis is more effective over the region of the round-window. In the postoperative management, an extremely important detail is to keep the scar on the promontory as thin as possible.

Patients, I have always found, are disappointed with results of operations on the mastoid if the discharge from the ear persists after operation, even when there is no odor to the discharge. In cases of chronic suppurative otitis media, if the disease is situated in the attic of the middle ear and mastoid antrum, and if examination and observation give evidence that the drum and ossicles are not involved and that the deftc into these regions is too small to permit satisfactory direct treatment, I prefer to use the modified radical operation for surgical interference. It is adequate for fulfillment of good surgical principles and, inaddition, permitting the drum to remain in place, it accomplishes two things: 1. it helps to conserve the hearing, and 2. it does not expose normal membranes on the promontory and in the Eustachian orifice, and thus prevents the occurrence of a wet ear. In my experience, unless the promontory is covered by scar, the ear is always moist.

It is gratifying to note in the current literature evidence of keen thinking and courage to express opinions about the problems that confront those of us who limit our endeavors to the field of diseases of the ear, nose and throat.

Dr. John R. Page (New York..: The surgical treatment of chronic mastoiditis is an interesting subject to discuss, and it is made particularly interesting by Dr. Smith because of his experience and skill. I am glad to hear him emphasize our obligation to develop different methods for different conditions, and to preserve or improve hearing whenever it is possible to do so with safety to the patient.

With respect to this and parallel with his idea of graduated technique is a plan of treatment that has been used in my clinic since I first took charge of it. It was published in an article on this subject in 1925, and it recommends first, as he does, in any treatment, the removal of tonsils and adenoids. It agrees with Dr. Smith that the type of operation should depend on the condition found within the mastoid and on the degree of hearing that is present, and that one should not follow the more radical procedure unless he is led to do so by the condition that is revealed on opening the mastoid. It recommends the careful consideration of the hearing and of the condition of the ossicles before the removal of any of them is contemplated, particularly where the hearing is good, for even when they are diseased it is not disease of the ossicles themselves that is the serious menace but the diseease that is around them.

In cases where there has been even great destruction of the drum membrane and marked changes in the tympanum, provided safety to the patient can be assured by such a measure, it recommends the "conservative mastoid operation," as Bondy calls it, or some modification of it to suit the individual case, and time for ventilation and treatment through a wide meatus to effect a satisfactory result.

I don't quite see why Dr. Smith is opposed to widening his approach to the tympanum by lowering the posterior wall of the canal and constructing a new meatus. It may be that he has had pockets form in the region of the mastoid tip that accumulated detritus, and that he obtained too large a cavity in the mastoid. But this can be avoided by removing the tip entirely and the ridge over the digrastic muscle so that the neck muscles fill up the space and lessen the size of the cavity, which then slopes downward and forward from the antrum to a wide meatus and is self-cleaning. In many cases the cortex can be beveled back so that the soft parts fall in so close to the inner plate the cavity formed has the same appearance as that after a simple mastoid operation that has a large canal to the drum. It seems to me that when the membranous and bony canal is left intact, as he describes, a narrower access to the part to be treated and poorer ventilation of the part will be obtained. Nor can I agree with him on removal of the incus alone to gain more space for irrigation and treatment of the diseased middle ear. With removal of the incus alone, it seems to me that the drum membrane acts more as an obstruction, or baffle, than an aid to hearing and that it might better be removed like the false membrane that sometimes forms across the tympanic part of a radical mastoid cavity, the incision or removal of which usually affords relief to the patient.

The old saying that when the hearing is poor it may he made better by a radical mastoidectomy, and when it is good it will be made worse is often true.

There has been a growing tendency in my clinic to attempt to preserve the drum and ossicles in cases where the hearing is too poor and the tympanum too much involved to warrant it. The radical mastoidectomy is more apt to improve the hearing and effect a dry tympanum in such cases than the modified radical.

This tendency is probably due to the fact that the treatment after a radical mastoidectomy is more painful and prolonged than after the conservative operation.

I congratulate Dr. Smith on his good work. I think that in his hands he gets results through a narrow canal that in other hands are more easily obtained through a wide one.

REPORT OF BOARD OF TRUSTEES OF THE CENTRAL BUREAU OF RESEARCH, AMERICAN OTOLOGICAL SOCIETY.*

DR. JOHN B. RAE, New York, and
DR. J. GORDON WILSON, Chicago.

The Annual Meeting was held in the New York Academy of Medicine on Jan. 8, 1938. The Morning Session was devoted to informal discussion, reports from all investigators receiving grants having been distributed to all members prior to the meeting.

Various requests for grants were considered. The increase in income was already accounted for by conditions of gift so that no new allocations could be made without encroaching upon investigations already under way. The Board was, therefore, regretfully obliged to refuse further grants meantime and the Chairman was requested to communicate this decision to applicants for grants.

At the Afternoon Session, Dr. Wesley C. Bowers, of New York, was presented to fill the vacancy caused by the untimely death of Dr. Arthur B. Duel.

Following discussion of heredity and environment in otosclerosis, the question of investigation of similar twins of known otosclerotic heredity was raised. It was hoped that the study of the onset and progress of the disease in these similar twins in varying environment would prove of value in our investigation.

It was determined to seek the aid of a geneticist as a Consulting Member of the Board to direct the method of attacking the problem, and the Chairman has since announced that Dr. Charles B. Davenport has kindly accepted the invitation and will so serve.

Dr. Wilson then read his report reviewing the work of the various investigators during the year. On motion, the thanks of the Board were extended to Dr. Wilson for his able presentation.

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*Read before the Seventy-first Annual Meeting of the American Otological Society, Inc., Atlantic City, May 6, 1938.

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REPORT OF THE OTOLOGICAL RESEARCH COMMITTEE.*

DR. J. GORDON WILSON, Chicago.

This report is concerned with studies done or in progress and may be conveniently divided into two parts: *1.* Anatomical and Pathological, comprising investigations of Prof. Bast, at Madison; Prof. Anson, at Northwestern; Dr. E. P. Fowler, Jr., and Dr. Jones, at New York. *2.* Physiological, investigations by Prof. Tait, at McGill; Prof. Davis and his associates, at Harvard; Prof. Culler, at Illinois; and Dr. E. P. Fowler.

1. Anatomical and Pathological: One outcome of these investigations has been to establish the wide anatomical variations which occur in the region between the stapes and cochlea, the seat of predilection for otosclerosis. So varied is the histology in this area that it is not possible to present an exact picture of a normal area in the fetus or infant, or even in the young child, other than in general terms. The most characteristic feature is a minute channel, found only in man, filled with connective tissue with a rim of cartilage surrounded by the bone of the otic capsule, called the *fissula ante fenestram.* The surrounding cartilage varies in thickness and extent, the bony trabeculae external to it contain marrow spaces of varying extent. In certain cases, not only in childhood but into adult life, masses of cartilage are seen at an age when the entire otic capsule is ossified. These occur most frequently: *1.* along the cochlear margin of the fissula; *2.* in connection with aberrant fissular extension above the tympanic opening; *3.* in the fissula at or near its tympanic opening; and *4.* where the fissula extends towards the stapes joint. This cartilage changes to atypical osseous tissue and it is possible that later an attempt will be made to resorb and reorganize such areas with resulting otosclerotic bone.

In the studies of the histological structure of the fissular area, it was noted that the cartilage of embryonic type is never wholly replaced by bone. It can become the source of newly-formed cartilage which obliterates the fissula and even

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protrudes from its tympanic orifice. Sclerotic masses may occur in close association with the cartilage, as if the one were the derivative of the other. Our continued observations, therefore, strengthen the opinion expressed in earlier publications, that the fissular area is histologically mobile, and that the persisting cartilage has potentialities of new growth and is associated with the production of sclerotic bone at this "site of predilection." An endeavor is being made to correlate the age period when such variations are most often observed with the period when bone formation is most active.

Dr. Bast continues his study of the utriculoendolymphatic fold and has published additional data to support his belief that this fold can act as a valve to close the utricular opening and so have an effect on pressure changes in the utricle. "From a purely anatomical point of view the utriculoendolymphatic valve should be capable of aiding in closing off the utricular opening of the utricular duct in all cases studied except in a few human types where there is present a variation of the valve."

Dr. Anson has recently published a further investigation of the anatomy of the duct-system, directed towards a better understanding of pressure changes in the membranous labyrinth, and shows that in the endolymphatic duct there are present structures capable of the physiological mechanism of absorption.

It has long been the desire of investigators of otosclerosis to be able to classify this local lesion in a recognized general pathological group. This has led to the study of various bone lesions which have attacked the petrous bone and, among others, Paget's disease. Dr. Anson and I have continued this study and have found that while the histological disturbances in Paget's disease are, in their essentials, similar to the characteristic changes elsewhere in the human skeleton, certain areas within the petrous pyramid resist the osteolytic processes destructive to long bones. While in the latter no part of the shaft or extremities is spared, in the temporal bone the otic capsule resists absorpton by osteoclasts. In one specimen (an adult, age 59 years), the trabeculae surrounding marrow spaces external to the osseous capsule were reduced to a mere lacework of spicules, while the osseous walls of the cochlea and semicircular canals were less affected. It would

appear that the cartilage islands are intrinsically resistant, that halisteresis is lessened, and important basic differences exist between these two diseases.

A consideration of the pathological processes which affect the petrous temporal has led to an examination of the routes by which infections may spread from the middle ear other than by the mastoid air cells. Since little anatomically is known of the air cells situated at the apex of the petrous pyramid, this subject has been studied by reconstructions, and one paper has been published and a second will appear this month. We find that these tubal air cells assume a fundamentally simple pattern. They appear as outpocketings of the mucous membrane on the medial tympanic wall, arising from the area just beneath the semicanal for the tensor tympani muscle, and the adjacent area near the inferior attachment of the tympanic membrane. From these sites they extend inward, within the bony wall of the carotid canal; some ascend, others are carried along the canal, the two groups of cells producing an almost complete loop of pneumatized tissues around the canal. The cells intercommunicate freely yet have no direct communication with epitympanic air cells of the mastoid area. The orifices by which the apical air cells connect with the tympanic cavity are undoubtedly the "fistulae" frequently seen at operation or at autopsy, not heretofore recognized as normal openings. Our initial study has been concerned with the anatomy of these tubal cells in a child, age 4½ years; we are now completing a further study in a child, age 13 years; other representative age-groups will follow.

The initial phase of the study of the otic capsule in the embryo has been published. Other stages will be carried out, all earlier than those described by Dr. Bast, and these several papers will account for the crucial stages in the development of the otic capsule.

Dr. Fowler, Jr., has correlated X-ray and pathological findings in 34 cases of inflammatory processes in the mastoid and petrous pyramid. In a detailed study of chronic otitis media his observations show that many cases are due to an actual invasion of marrow spaces in the temporal bone instead of an invasion in the air spaces as generally believed; long-standing and recurrent discharges from the middle ear

may be due to an osteomyelitis rather than simple osteitis. He has studied with audiograms, X-ray and serial sections a case of multiple myeloma of the temporal bone, considered clinically to be osteolytic.

Physiology: Considerable investigation has been directed to determine the relation of the normal perception of sounds by air or bone to the electrical response generated in the cochlea by the Wever and Bray method. Since the electrical or cochlear response seems a promising approach to estimate hearing in experimental animals, its relation to normal audition, dependent on the integrity of the organ of Corti and the VIIIth nerve, is a fundamental problem to be settled before one can proceed to correlate experimental findings to clinical results. At present, the views of physiologists seem sharply divided: In one group, we find Prof. Bast and Prof. Tait; in the other, Prof. Davis and, with reservations, Prof. Culler.

Dr. Bast expresses his views briefly and emphatically: "The development of cochlear potentials in response to air- or bone-conducted stimuli does not depend on the structural integrity of the organs of Corti or associated structures; the hair cells and nerve elements in the cochlea are not essential for the production of cochlear response." With this opinion, Prof. Tait is in general agreement, as also are a distinguished group of English experimental physiologists. The English investigators regard normal hearing (nerve tract potentials) as resulting from the vibration of a resonant structure (basilar membrane); on the other hand, cochlear potentials come from the vibration of a nonresonant, highly damped structure, and the hair cells and nerve elements associated with this structure are not essential for their production. Following section of the VIIIth nerve in cats, pathological changes occur in the internal ear, yet normal cochlear responses were obtained in several animals in which there were extensive degenerative changes and disintegration of neural elements and of the organs of Corti.

Contrast this with Dr. Davis' opinion: "For the present, we adhere to our view that both normal hearing and the electrical activity of the cochlea depend upon the integrity of the organ of Corti." To explain his findings in a normal animal, whose conditional reflexes did not correspond with the cochlear response, he suggests there is a limitation of

the method of conditional reflexes, which appears due to idiosyncrasies of individual animals, or it may be of actual partial deafness, depending upon some undetected lesion of the higher nervous pathways. "Within these limitations, however, we believe that the results obtained by the electrical method of testing fairly represent the true physiological performance of the ear, and that abnormalities of the threshold for electrical activity may be understood in detail in terms of anatomical abnormalities. We base this conclusion, primarily, on the correspondence between local damage to or survival of the organ of Corti and tonal gaps or islands. It is obvious, however, that the agreement is not perfect in all details, and we must consider the observations of Bast and Eyster and of the English investigators, Ashcroft, Hallpike and Rawden-Smith. These investigators do not find any clear correlation between electrical activity and the condition of the organ of Corti, and the British group ascribe the origin of the electrical phenomena to vibration of electrically polarized membranes. We are at present unable to explain all of their observations in terms of our view." These investigations are being continued in collaboration with Dr. Dworkin.

Closely related to these views on cochlear potentials is the question of localization of frequencies in the cochlea. Believing in such localization, Dr. Culler has prepared a map of the localization of pitch in the cochlea of the guinea pig, based upon a survey of cochlear responses. In conditioned animals, he checked electrical potentials with hearing tests, in which localized cochlear lesions were done at points marked *200* and *250* cycles on his map. The following results were obtained in seven guinea pigs: *1.* The maximal hearing loss is around 200 cycles in two cases; 350 in four; and 300 in one. This agreement with electrical map appears good, though not quite perfect. *2.* The loss extends over a considerable range; all of the test frequencies (125 to 1,000 cycles) are impaired by the lesion; the spread is widest directly after operation; as the perforation closes, the loss begins to focus about one point. *3.* From the maximum, the loss spreads through lower frequencies more than higher, as though the oscillations coming from the oval window were specially attenuated for all frequencies beyond the perforation.

Contrast with this the report of Prof. Tait. He made small circumscribed lesions in different parts of the cochlea of

cats and studied the subsequent functional and histological changes. He finds total deafness in some cases; in others, the loss of hearing is partial, but is not confined to any one frequency or restricted group of frequencies. He is continuing these investigations in collaboration with the Harvard group.

It is obviously desirable to improve the auditory deficiency tests now in use, to determine very early spread of the otosclerotic lesion leading to fixation of the stapes. Dr. Fowler, Sr., has suggested an approach, called by him the "hobble" effect. It is unnecessary to dwell on this, since Dr. Fowler has on several occasions explained this method. Though still in its experimental stages, one hopes that it will justify reports already made and prove an important aid to the early recognition of stapes fixation.

In the spring of 1937, I spent two days in the laboratory of Dr. Knudsen and his assistants in Los Angeles, and was impressed with the enthusiasm and care with which the work was carried on. They have an elaborate set-up directed to the investigation of the selective amplification best suited to different types of impaired hearing.

Prof. Collip, aided by Dr. Mortimer and Dr. Wright, is making a preliminary study of the effect of a selective group of endocrines on auditory acuity.

Your Committee proposes to investigate the influence of environment on otosclerosis, in subjects whose family histories point to an otosclerotic susceptibility. Not unaware of the difficulties involved in such an investigation, we have been fortunate in securing the aid of Dr. Davenport, of the Department of Genetics, Carnegie Institute, Washington, in this phase of our work.

From the very satisfactory reports presented, I believe we have the right to congratulate ourselves on the progress being made and I ask that you permit me to tender our thanks to those receiving grants and our continued interest in the progress of their investigations.

104 S. Michigan Avenue.

REPORT OF THE CHICAGO COMMITTEE ON OTOGENIC MENINGITIS.*

DR. ALFRED LEWY, Chicago.

In last year's report we were somewhat enthusiastic about results in otogenic hemolyticus meningitis cases treated by surgery, plus sulfanilamide. Gradually, we are learning more about the pharmacology of this drug and its clinical application.

A survey of five cases, briefly described below, leads to a better definition of our views as to what role surgery should play in the management of these cases.

Case 1: Courtesy of Dr. F. J. Cooper and Dr. W. J. Noonan. Boy, age 7 years. Streptococcus hemolyticus meningitis. Positive culture: Hemoglobin, 56 per cent; reds, 3,270,000; leukocytes, 12,800. Mastoidectomy; sulfanilamide by mouth, 5 gr. every four hours; intramuscularly, 5 cc. every eight hours. After every large dose a condition of somnolence and cyanosis. At the end of two weeks, no effect on temperature, spinal fluid or neurological symptoms. Lowest hemoglobin, 38 per cent; red cells, 2,640,000. Then 0.8 per cent sulfanilamide given intraspinally, followed by clearing of formerly turbid fluid, cell reduction from 3,480 to 605, sterile culture and recovery.

This case raises the question of what constitutes the optimum concentration of sulfanilamide in the spinal fluid. We have tentatively accepted 10 mils per 100 cc. as the optimum for the blood stream. The question should be answered if possible.

Case 2: L. W., age 11 months. Admitted to Contagious Hospital in poor condition, Jan. 27, 1938. Diagnosis: postmeasles bronchopneumonia. Neck rigidity, Brudzinski, ears negative or doubtful. Spinal fluid culture, streptococcus hemolyticus. No operation. Sulfanilamide therapy. Recovery.

Case 3: Courtesy of Dr. Samuel Pearlman. W. S., age 11 years, entered Cook County Contagious Hospital, Oct. 11, 1937. Head cold two and one-half weeks previously. Struck on head by football 11 days previously. No unconsciousness or bleeding from ear, nose or throat. Headache and earache, 11 days. Vomiting, five days. Stiff neck, two days. Temperature, 105.5.° Spinal fluid, 3,000 cells. Pandy, 4+. Culture, abundant growth, hemolytic streptococci. When seen by Ear, Nose and Throat Department, bulging left tympanic membrane was disclosed. On paracentesis, large amount of pus was obtained. Schwabach, prolonged; Weber, to left; whisper, one foot left. No mastoid tenderness; skull and mastoid negative for fracture or cell destruction; no edema of optic disks. No operation. Patient received ¾ gr. sulfanilamide per pound body weight

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(60 gr. daily) for three days. Pleocytosis diminished gradually but persisted over 40, predominately lymphocytes. Course essentially afebrile after 48 hours. Hearing returned to normal. Neurological examination negative on dismissal, and he has remained well.

Case 4: Courtesy of Dr. Frank J. Piszkiewicz. A male infant, age 20 months, seen October, 1937. Upper respiratory infection of three days' duration, followed by bilateral otitis media. Paracentesis; pus obtained under pressure from each ear. At this time the infant was acutely ill, with neck rigidity; positive Kernig and Brudzinski, and Babinski, right. Spinal fluid under pressure, cloudy, 17,000 cells per cubic millimeter; smear and culture, hemolytic streptococcus. The following day, paralysis of the right external rectus. Operation refused by parents. Clinical cure in three weeks.

Case 5: Courtesy of Dr. Joseph E. Caliendo. M. P., female, age 16 years. Admitted West Side Hospital, Feb. 24, 1938. Two weeks ago, ear discharge of two days' duration. One week ago, vertigo and vomiting, sweats, followed by headache. Lost 12 pounds in two weeks. On admission, temperature, 103°; pulse, 112; respiration, 26. Blood pressure, 120/80. Hemoglobin, 60; red cells, 3,950,000; whites, 35,600 stiff neck; Kernig; Brudzinski present; knee jerks; abdominal reflexes absent. X-ray of mastoid; doubtful clouding, left. Paracentesis brought only few drops pure blood, smear and culture of which, negative. Spinal fluid turbid, under pressure, 650 cells. Culture, streptococcus hemolyticus. Seen by me, Feb. 26, 1938. Dysmetria, left hand; spontaneous horizontal nystagmus, both directions; membrana tympani, left, no redness or swelling; blood clot adherent to membrane. Hearing for whisper, present both ears. Caloric reaction absent, left; hyperirritable, right. Two consultants disagreed as to whether surgery was advisable. I believed the symptoms were accounted for by meningoencephalitis, with the ear as a causative factor doubtful, or in any event without any localized lesion of the ear or mastoid in the surgical sense, and cast the deciding vote against operation. Patient made a good recovery, and examination, April 18, 1938, showed no remnant of neurological disturbance, and hearing is normal in both ears.

Our statistical record of a 97 per cent + mortality in otogenous hemolytic streptococcus meningitis, when treated by surgery and supportive treatment alone, indicates that the surgery, *per se*, did not avail much; therefore, I conclude that in hyperacute cases of otitis media with hemolytic streptococcus shown in culture of the spinal fluid, the meningitis occurring so early that evidence of breaking down or localization in the mastoid is lacking, and in which the otitis may be coincidental, we are justified in withholding surgery beyond paracentesis, and trust to sulfanilamide plus supportive treatment. Meningitis occurring in chronic suppurative otitis is another matter.

25 East Washington Street.

REPORT OF THE AMERICAN OTOLOGICAL SOCIETY REPRESENTATIVES ON THE COMMITTEE FOR HEARING AIDS AND AUDIOMETERS OF THE COUNCIL ON PHYSICAL THERAPY OF THE AMERICAN MEDICAL ASSOCIATION.*

DR. EDMUND PRINCE FOWLER, New York.

Your representatives, Dr. Newhart and myself, have attended with one exception all the meetings of the Committee on Hearing Aids and Audiometers. The Council on Physical Therapy has authorized publication of the following report. Instead of reading this report, I shall distribute one to each of you and add thereto some brief comments, which it is hoped will serve to clarify it.

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COUNCIL ON PHYSICAL THERAPY.

The Council on Physical Therapy has authorized publication of the following report:

The Council wishes to express its appreciation for the value of assistance rendered in the preparation of this report by Dr. George M. Coates, Dr. Lee W. Dean, Dr. Edmund P. Fowler, Dr. Austin A. Hayden, Dr. Isaac H. Jones, Dr. Douglas Macfarlan, Dr. Horace Newhart, Dr. Burt R. Shurly and Dr. William P. Wherry.

Howard A. Carter, Secretary.

PURPOSE OF AUDIOMETERS.

A clinical audiometer is an instrument for measuring the acuity and range of hearing.

TENTATIVE MINIMUM REQUIREMENTS FOR ACCEPTABLE AUDIOMETERS.

A. Specifications:

1. Audiometers shall produce vibration at frequencies within hearing range (approximately 128 to 8,192 cycles, or higher, per second). They shall be equipped for testing both air and bone conduction.

2. Frequencies: a. Continuous frequencies (sweep) from 128 to 8,192 (or higher, for example, to 16,000) cycles per second.

b. Fixed frequencies from 128 to 8,192 cycles per second. If discrete frequency steps are provided, the tones shall be 128, 256, 512, 1,024, 2,048, 4,096, 8,192 cycles per second. Numerical annotations to be used designate pitch.

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c. The limits of tolerable frequency variation shall be not more than ±5 per cent at all frequencies. Dials shall be marked so that frequencies may be identified readily.

3. Attenuation: Audiometers shall be calibrated in decibels, with 5 dcb. per step or less. In no case should more than 5 dcb. steps integrals be used. Toleration limits to be within ±1½ dcb. per 5 dcb. steps and ±5 dcb. cumulative at any portion of the intensity range. Dials shall be easily read. The term "percentage hearing" shall not be used, but hearing losses shall be reported in decibels units loss.

4. Range of Intensity. The intensity range of the test tones above the normal threshold shall be at least that as follows:

Test Tone Cycles per Second	Intensity Range Decibels
128	60
256	80
512	85
1,024	90
2,048	90
4,096	90
8,192	70

5. Wave Form: The purity of the tone in the air conduction receiver shall be such that the harmonics at any particular frequency shall be at a level not less than 25 dcb. below the fundamental tone or that other accessory noises (such as the line hum, click of interrupting switches, etc.) shall in any way interfere with the test tone.

6. For a bone conduction receiver, the sensation level of the sound reaching the tympanum through the auditory meatus shall be at least 5 dcb. below the level generated by bone conduction at all test frequencies as judged by a normal ear, when the bone conduction receiver is placed on the mastoid region.

7. Power Supply: Either alternating or direct current, alternating-direct current or battery.

8. Ruggedness of construction to stand reasonable usage. Use of readily obtainable and replaceable parts.

9. Uniformity in calibration in decibels.

10. Advisability of the selection of a central, distinterested agency for reporting on physical characteristics of various audiometers.

11. Advisability of manufacturers assuring servicing.

B. Audiogram or Auditory Chart:

(To be adopted later when a more definite consensus of opinion develops.)

C. Definition of Threshold of Hearing: The threshold of hearing is the audiometer setting corresponding to the lowest intensity at which the person being tested is able to indicate correctly more than half the time that he is hearing.

D. Marketing and Advertising: Rules of the Council on Physical Therapy shall be adhered to by manufacturers of acceptable audiometers.

COMMENTS.

A. 1. A cycle is a double vibration.

2. (c) This means that there may be a variation of five cycles to either side of the true designated setting on the frequency dial.

3. A decibel may be described as approximately the minimum perceptible change in intensity that can be sensed as such.

The toleration limits prevent unequal or excessive jumps between dial points. In other words, it insures a smooth control of the intensity steps.

4. The intensity range for 8,192 was changed, subsequent to the printing of the original report, from 80 to 70 dcb.

5. In other words, the first overtone and all subsequent overtones shall be at least 25 dcb. (40 dcb., as originally printed, is a clerical error) fainter than the fundamental at any and all intensities and frequencies.

6. This is possible only with proper technique in application.

B. Avoid complicated and tricky machines and charts and charting methods. They often serve only to cover up defects in calibration of frequencies and intensities. The graphs may be meaningless, as compared to standard audiograms. They tend to discredit audiometric methods.

C. The possibility of thus determining the threshold is peculiar to the audiometer. It cannot be accomplished with anything like precision by tuning forks.

A meeting has been called for Aug. 6, 1938, to consider particularly the minimum standards for hearing aids, and the ethics of manufacturers and sales agencies. Few otologists understand the recent improvements in electrical hearing aids and the difficulties involved in manufacture and fitting. They, as well as patients, often expect the aid to take the place of a normal ear; this it can never do. In the present stage of rapid advances in machines and technique it is impossible to lay down final or ideal standards; however, from year to year, standards will be raised as science permits.

PART II.

DR. HORACE NEWHART, Minneapolis.

Supplementary to Dr. Fowler's concise presentation, I would make a few statements in answer to some of the many inquiries recently made by members of this Society and others. This can be most expeditiously done by question and answer.

Question 1: Who constitute the Committee of Consulting Otologists?

Answer: Nine interested members of the American Otological Society, whose names appear on the reprint being distributed. The Chairman is Dr. Austin A. Hayden, who, because of his interest in better hearing, was inspired to interest the American Medical Association Council on Physical Therapy to undertake the task of standardizing audiometers and insuring the production and ethical distribution of high-grade electrical hearing devices.

Question 2: Why has the Committee, after releasing its statement of tentative specifications, failed thus far to recommend to the Council on Physical Therapy, *as acceptable* any of the several audiometers submitted to it? Such delay has been seriously disappointing to prospective purchasers and manufacturers.

Answer: Because of unexpected technical difficulties encountered in the solution of the problem of formulating specifications for receivers of standardized performance, which is a fundamental need for producing audiometers capable of yielding uniformly comparable results.

Question 3: Which audiometers of those now on the market do you personally recommend as the best?

Answer: None. As a member of the Committee, one cannot consistently recommend any one instrument to the exclusion of others. There are available today several instruments, each of which have different features of real merit and equipped with various accessories, such as bone conduction receivers, different types of masking devices, built-in microphones, etc., some of which are essential for accurate diagnosis and for the selection or prescribing of hearing aids. To

the otologist in urgent need of an audiometer for diagnostic purposes we would suggest that after careful consideration of his needs, he purchase the instrument which will best meet his requirements. In doing so, he should demand of the vendor that in case the instrument should not meet the specifications soon to be released and, within a reasonable time after purchase, he will guarantee for a reasonable consideration to make good by exchange, substitution of parts or remodeling. Such terms are recognized as just and reasonable by several makers, so great is their confidence in the high grade of their products.

Question 4: When will the problem of a standardized receiver be solved so that acceptable audiometers can be manufactured?

Answer: We do not know, but sincerely hope in the very near future. Undue haste would defeat the objectives of the Committee. As a member of this Committee, I am grateful to the Council on Physical Therapy of the American Medical Association for its generous, effective work in promoting higher standards of service in otologic practice.

There is a need for at least two types of pure tone audiometers, in addition to one for ordinary diagnostic purposes:

a. An instrument designed especially for research and use in dealing with the severely deafened. Such an audiometer will afford a wider operative range with respect to both pitch and frequency and will make available pure tones in continuous band, in addition to selection at fixed intervals.

b. A simple, reliable pure tone audiometer at low cost for rapidly detecting and accurately measuring hearing loss in individuals of large groups by air conduction only. Such audiometer should have a range of 128 d.v. to 8,192 d.v., inclusive, and be built to meet the minimum specifications of the Council on Physical Therapy. An instrument of this type in no sense should be used for diagnostic purposes.

THE AMERICAN BOARD OF OTOLARYNGOLOGY.*

DR. WILLIAM P. WHERRY, Omaha.

American Otological Society;

American Laryngological Association;

American Laryngological, Rhinological and Otological Society;

American Academy of Ophthalmology and Otolaryngology;

Section on Laryngology, Otology and Rhinology of the American Medical Association.

Herewith is report on examinations given by the American Board of Otolaryngology at examinations held during 1937:

City	Total Exam.	Passed	Formal	Certified	Conditioned	Pending
Philadelphia	97	67	7	74	23	.....
Chicago	103	68	12	80	22	1

Total otolaryngologists certified to date, 2668.

1500 Medical Arts Building.

*Read before the Seventy-first Annual Meeting of the American Otological Society, Inc., Atlantic City, May 6, 1938.

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MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

SECTION OF OTO-LARYNGOLOGY.

Meeting of Oct. 8, 1937.

Laryngeal Obstruction. Dr. F. D. Hurd.

(Continued from June issue.)

DISCUSSION.

DR. LAWRENCE R. BOIES: I am sorry Dr. Hurd did not take the time to read the original paper prepared for the staff meeting at the University Hospital. It was much more complete than his discussion of this evening.

The choice of method in relieving obstruction merits some discussion. Mention was made of the fact that the cases seen at the University do not include many of the type seen in a hospital with a busy contagion service.

When the cause of obstruction is a tumor, paralysis, a congenital anomaly or a chronic infection producing stenosis, tracheotomy is the unquestioned procedure in view of the prolonged treatment necessary. To relieve an obstruction due to an acute inflammation, the edema which occasionally results after instrumentation, an allergic reaction, etc., intubation has a distinct usage, but there are definite situations in which tracheotomy is the procedure indicated.

The following considerations are important: 1. Intubation is a relatively simple procedure, quickly accomplished in the hands of an experienced operator. However, it demands that an experienced intubator be quickly available to put the tube back once it is coughed out. 2. Repeated intubations or prolonged use of an intubation tube produces an irritation in the subglottic area which may cause stenosis. "Tracheotomy conserves the laryngeal structure better than intubation in infants." (Tucker.) 3. An infant will not take food or fluid by mouth satisfactorily with an intubation tube in place. 4. In the presence of an infection producing a membrane below the glottis, as in an acute laryngotracheobronchitis, an intubation tube is inadequate. A tracheotomy, in addition to providing an adequate airway, facilitates removal of the inspissated secretions and offers drainage. The factor of drainage provided by tracheotomy in the acute fulminating infections has probably been overlooked. 5. The fact that a tracheotomy opening does not admit air warmed and moistened in the upper respiratory tract has been shown from clinical experience to be an unimportant consideration. The use of a warm steam room is a satisfactory substitute.

The old descriptive terms of a high, median or low tracheotomy have been discarded. All tracheotomies should be low except those preliminary to laryngectomy when conservation of as much of the trachea as is possible is important.

Tracheotomies may be conveniently classified as "emergency" or "orderly"; emergency tracheotomies may still be orderly in cases of extreme dyspnea. The use of a bronchoscope or Mosher life-saving tube inserted through the glottis changes the procedure from an acute emergency to an orderly one and produces a more satisfactory and a safer technical effort.

We prefer the removal of a disc of cartilage slightly larger than the tube to be inserted. The incision, unless unusually long, is not sutured. Drainage around the tube is important and suturing causes more reaction and a tendency to emphysema. Experimental work (Richards and Glenn) has shown that this type of opening does not interfere with the patency of the tracheal

lumen after the tube is removed and healing has taken place. It seems illogical to insert a tube through a narrow longitudinal or transverse slit with the resultant tension on the margins of this slit. An adequate opening facilitates exchange of tubes with the least amount of irritation. It also facilitates the matter of drainage from the trachea.

There seems to be a tendency to use too small a tube. Clinical observation indicates that the size of the lumen is not a factor in producing irritation in the trachea. In this respect, only the length of the tube is important. The larger the lumen of the tube, the less the tendency for it to clog with mucous to an extent to interfere with an adequate airway. A larger lumen is also easier to keep clean.

Nursing care of tracheotomized patients by those experienced in the management of this type of care is extremely important.

Tracheotomy wounds tend to heal promptly without surgical interference, even though tubes have been worn for months.

Patients are surprisingly comfortable in permanent tracheotomy. Tracheitis and bronchitis are uncommon after the variable amount of this reaction present in the first few days after the operation. There seems to be no increased susceptibility to pneumonia. Thomson and Wood have each reported a case of tracheotomy tube worn over 70 years. Wood remarked that his patient claimed that she had never had bronchitis.

DR. VIRGIL J. SCHWARTZ: At the Minneapolis General Hospital, we have had considerable opportunity to study acute laryngeal, tracheal and bronchial infections. Many were diphtheritic, but others were streptococcic, and a number appeared as a part of the general mucosal inflammation of scarlet fever. Most of you are familiar with the technique, developed in the East some years ago, for the treatment of accumulations of diphtheritic membrane in the trachea by suction. This has been so successful that in many instances it has done away with the necessity for intubation or tracheotomy. Scarlet fever or streptococcic cases are apt to give as much trouble as, or more than, the diphtheria cases in some instances. Occasionally, there is so much edema and infiltration of the mucosa of the larynx and trachea, and it extends so far into the bronchi that even tracheotomy does not help, since the mucosal walls below the cannula are almost in apposition because of the swelling. In addition, this mucosa is very hemorrhagic, so that bleeding from the slightest irritations, such as the presence of a well fitting cannula, is difficult to control.

I think that both Dr. Hurd and Dr. Boies will agree that probably one of the most common causes of death after tracheotomy is not the tracheotomy itself, but the fact that it was postponed too long and the patient really dies of exhaustion.

The point was brought out that intubation itself may produce laryngeal stenosis. I think this is a very important point, and one which I have emphasized for some years. It should be remembered that when the mucosa of the larynx and trachea is inflamed, often a tightly fitting intubation tube only adds fuel to the fire, producing more swelling, and unless we are extremely careful stenosis may follow. This happened in a case which was transferred from the General to the University Hospital. This child had been intubated several times, and subglottic stenosis developed immediately beneath the vocal cords. A tracheotomy was done, but, as might be expected, decannulization was not possible because of the stenosis.

(To be continued in a succeeding issue.)